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The Bancroft Memorial Lecture.[1]

SOME ASPECTS OF INTRACRANIAL SURGERY, WITH SPECIAL REFERENCE TO THE MENINGIOMATA.

By HAROLD R. DEW, F.R.C.S.,
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THIS lecture has been founded to commemorate the work and influence of a remarkable doctor, Joseph Bancroft. A memorial lecture must be acknowledged to be one of the most fitting ways of perpetuating the memory and influence of a notable person, and I am very appreciative of the honour done me in your asking me to deliver this lecture, although I am only too conscious of the burden it has laid upon me—not only to do the occasion justice, but to follow adequately those distinguished lecturers who have preceded me. It is customary on these occasions to speak about the man whose name the lecture takes, although doubtless all here present know as much, if not more, about Bancroft than I do. Joseph Bancroft, who had such a wide range of interests and whose contributions to a variety of applied scientific subjects were so numerous, represents a type of doctor that was much more common during the last century than it is today, the doctor-naturalist, one imbued with a great curiosity about all natural phenomena. Our profession, luckily, has always numbered some men of this type in its ranks. They were men who were very often mentally far in advance of their times, and who, by their freedom of thought and action, had a stimulating influence on their contemporaries. Bancroft, even for his contribution to the filaria story alone, earned world-wide recognition, and he

[1] Delivered at Brisbane on June 5, 1936.

is among the few Australian doctors whose names will never be forgotten.

This type of doctor was able to take an interest in all sorts of biological problems, mainly because it was then possible—owing to the rudimentary knowledge of much of what we call modern medicine—for a brilliant man to know practically all that was worth while and still have time for other interests. Owing to the enormous growth of medical knowledge that day has almost passed, and it is now practically impossible for an individual to know completely more than a small part of medicine. This has led to the development of more or less narrow specialization, which, whatever its disadvantages may be, has undoubtedly led to wonderful advances in our knowledge, advances which could never have occurred had not workers concentrated their attention on a relatively limited field. We are not yet finished with surgical specialization, as we are now seeing the rise of specialization in thoracic surgery, surgery of the rectum, and neuro-surgery; it is possible that in the future there may be a more important development, already illustrated by the activity of Brunn, Sauerbruch, Davies, and to some extent of Cushing, in which the specialist is both physician and surgeon and deals with all diseases affecting the region in which he has specialized. Few, if any, of these special branches were established in Bancroft's time, but I feel sure that he was the type of man who could see these changes coming and who, had he lived a little longer, would have played no small part in their evolution.

It is of one of these recent specialities, that of neuro-surgery, with special consideration of one type of intracranial tumour, that I desire to speak tonight. It has often been said that the car of progress travels forward on square wheels, very slowly and with many a stop and many a jolt. That is particularly true of the surgery of the central nervous system. Up to quite recent times the successful removal of a brain tumour was a rare event, and although odd successful cases were recorded even in the early part of the century, no great advances comparable with those in other regions took place in this field. The reasons were not far to seek. Knowledge of the function of the central nervous system was sadly lacking and had to be built up by men like Ferrier, Broca, Jackson, and by the spade work of numerous others. At the same time, knowledge of clinical neurology was poor, until the British school of neurologists in particular, with Jackson, Gower, Mott and others, paved the way. Our knowledge of the physiology of the brain, and the cerebro-spinal fluid, too, was slight until the rise of experimental methods solved many of the problems. This work is, of course, associated, to mention but a few, with the names of Horsley, Sherrington and Holmes in England, and of Starr, Cushing, and Weed in America. Still, even until 1930, very little progress had been made in the surgical attack on brain tumours. Further knowledge of methods of localization, of pathological processes and of technical methods was needed if we were to convert the occasional into a more or less uniform success and to place the subject on a sound scientific basis.

A hopeless attitude too was often engendered amongst physicians and surgeons because of the fatalities and the apparently hopeless outlook in the majority of cases. Diagnosis was often not made until too late because of the lack of interest, while many cases were allowed to become hopeless before any surgical attack was attempted. It was obvious that education of the profession and public concerning the advances that were being made and the possibilities of the future was badly needed if any improvement in results was to be achieved. The story of the last two decades provides ample proof of the soundness of the principle that more rapid progress is made in the solution of any clinical problem if it can be attacked in all its aspects by a group of skilled workers.

In cases such as these, many independent experts were often called in to help, but in spite of this progress had to be uncertain and haphazard. The neurologist alone is handicapped because he has not the often illuminating experience of testing his diagnosis on the operating table; the surgeon, who is sometimes almost entirely interested in matters of technique, tends to throw the onus of a mistaken diagnosis on the neurologist and to lose interest in the case, while the pathologist, often working on small pieces of tissue, loses much if he has not seen the lesion *in situ*. It was left to Cushing to collect a team of workers which attacked every aspect of intracranial surgery, and which, in a series of illuminating papers and monographs, established the surgery of the central nervous system, both neoplastic and traumatic, on a correct basis. In addition, trained observers and workers have been sent out into numerous centres in various parts of the world and have there done much to advance this type of work.

So much has the subject advanced during the past fifteen years that there is already a voluminous literature in all languages, and papers a few years old are already out of date. This has been accompanied by a remarkable increase in our knowledge, not only of the pathology of these lesions, but of the clinical aspects, so that new syndromes are continually being differentiated, while new additions to the operative armamentarium have revolutionized both diagnosis and treatment. At present, indeed, the operative treatment of tumours and other lesions, instead of lagging behind, is tending to run in advance of our diagnosis and understanding of clinical phenomena.

All this has been a somewhat intermittent development; its story and that of the contributions made by various workers would take up many lectures and need not concern us here. I should like to point out, however, that the specialty is well established, and the results, both as regards advances in our knowledge and improvement in treatment, have been little short of miraculous.

In considering intracranial tumours generally, we have three methods of approach. We can consider tumours of certain histogenetic types wherever they occur in the brain; tumours of various types in a special site—a purely regional attack which was the common method in the past; or tumours of a particular type in a special site. The last method has been used by Cushing with conspicuous success in his work on the tumours of the eighth nerve and of the pituitary gland. The modern tendency has been to deal with tumours of the same histogenesis because on this histogenesis their pathological nature and behaviour and very often their site have been found largely to depend. Besides, it is of the greatest possible value for the surgeon to know beforehand the type of tumour with which he has to deal; on this depends not only the prognosis, but very often the operative technique to be used. Thus it is more than disconcerting for a surgeon to encounter in the course of what he expects to be a simple operation, such as a decompression, a vascular meningeal tumour, and so to have to face an impromptu, hazardous and bloody performance, particularly if he has omitted to provide himself with muscle for hæmostasis, with a blood donor, or with a reliable diathermy machine.

I intend to adhere to the first method of attack and to choose for general review out of the whole range of neuro-surgery a specific type of tumour, the meningioma — an essentially benign tumour derived from the meninges.

THE MENINGIOMATA.

Historical.

These interesting tumours have masqueraded under so many different names that it is difficult to deal with them adequately from the historical point of view. Cruveilhier,[1] in 1835, recorded four examples of dural growths, which he called *"Tumeurs cancéreuses des méninges"*. Paget[2] called them myeloid tumours because of their association with bone invasion, while in 1859 Virchow[3] adopted the term "psammona" for those which contained calcareous particles, although later he classified some of them as sarcoma of the *dura mater*. As aniline dyes and improved histological methods were adopted, their microscopic study became easier, but, as usual, purely histological considerations assumed undue importance and a great variety of descriptive names was given to them. Golgi[4] and others popularized the term endothelioma, on the assumption that the lesions came from dural mesothelium; but the term is inapplicable because these tumours rarely, if ever, arise from the *dura mater* itself. In an attempt to overcome some of the difficulties of nomenclature, Cushing,[5] about 1922, introduced the term meningioma, to include the entire group of tumours which appear to arise from the *dura mater*, whether mesothelial or fibrous elements predominate, and no matter what the general appearance. It will be seen later that even this name is not an ideal one, but it is relatively non-committal and has come to be more or less universally employed.

Histogenesis.

In 1864, Cleland[6] pointed out how the type cells of these neoplasms closely resembled the cells of the Pacchionian granulations of the arachnoid and suggested that most, if not all, of these tumours were derived from these structures. Many later observers agreed with this, and Schmidt[7] (1902) demonstrated the common occurrence of clusters of similar cells between the layers of the *dura mater*, particularly with advancing age. He recognized that these were often associated with arachnoidal tufts and occurred rather often in the same sites.

The normal arachnoid villi consist of a connective tissue stroma at the apex of which is a cluster of arachnoidal mesothelial cells, and some of the tumours in question might quite readily be taken for a grossly hypertrophied Pacchionian granulation. Casper[8] has clearly shown transitions between the arachnoid cell clusters and the meningeal tumours in three cases of multiple meningiomata.

It has also been pointed out by Benecke,[9] Schmidt, and Cushing[5] that in many cases of raised intracranial pressure minute cerebral herniations occur through defects in the *dura mater* which are normally the sites of arachnoidal tufts, and it is found that the sites of these tufts more or less correspond with the distribution of meningeal tumours.

Weed,[10] in 1930, demonstrated the frequent occurrence of similar cell clusters in the meninges in elderly cats. These clusters showed a similar variability in structure to the tumours in man.

The studies of Weed[11] and others have shown very clearly that the arachnoid tufts normally act as organic filters in the path of the cerebro-spinal fluid circulation between the subarachnoid space and the dural venous sinuses. It is possible that, following injury of any sort, these may become temporarily blocked by *débris* and necrotic cells and that this may stimulate the arachnoidal cells to phagocytic activity and also stimulate them to multiply. It is possible that such an hypertrophy may persist under certain conditions and so be the genesis of neoplastic formation. It is obvious that as age advances there is more chance of these changes taking place, and there seems every possibility of actual neoplastic change sometimes occurring in one of these areas. This may be, then, a possible explanation of the frequency of a history of severe trauma in patients with these meningeal neoplasms.

Numerous studies of the development of the membranes of the brain have been made in recent years. Weed, from his work[12] with pig embryos (and practically all other observers agree), believes that the *dura mater* and the arachnoid differentiate from each other through the early condensation of groups of cells in the perimedullary mesenchyme and not that the outer condensation is the precursor of *dura mater* alone. This layering is caused by the passage of cerebro-spinal fluid which is relatively early elaborated in the region of the

fourth ventricle and makes its way between the groups of mesenchymal cells. Harvey and Burr,[13] using transplantation experiments on amphibian larvæ, however, came to the conclusion that the *dura mater* alone was developed from the mesenchyme around the neural tube and that the leptomeninges were developed from the neural crest and so were epiblastic in origin. The work of Harrison along similar lines supports the view that the sheath of peripheral nerves, a somewhat analogous structure, may have an origin from the neural crest and, as is well known, this has been seized upon by Masson and others to support the epiblastic origin of such tumours as the schwannomata.

Further evidence of the mesodermal origin of these tumours has been brought forward by Buckley and Eisenhardt,[14] who have studied tissue cultures of a parasagittal meningioma. They found that the cells grew in the medium in a reticular pattern as do cells of mesodermal type, although they stated that as the cells did not lay down collagen, they did not, in their opinion, resemble fibroblastic cells.

Elsberg[15] has pointed out that in tumours of this type, affecting the spinal meninges, the arachnoidal origin is not always easy to prove. Tumours have been found actaully outside the *dura mater* as well as inside it, but entirely free from the cord itself.

It would seem, however, that during the differentiation of the layers of the meninges ample opportunity would exist for some of the cells destined to be arachnoidal to become left behind as cell rests at various levels, and that from these scattered cell rests tumours of arachnoidal type might arise.

The consensus of opinion, however, seems to favour the view that all the meninges are truly mesoblastic structures, so that histogenetically all tumours having origin therefrom must belong to the connective tissue and not to the epiblastic series. This is strongly supported by Mallory[16] and Penfield,[17] who believe that the type cell is the fibroblast, basing their conclusions on the production of collagen and fibroglia by the tumour cell. Mallory has introduced the term arachnoid fibroblastoma to emphasize the origin from arachnoidal cells, wherever the tumours may appear to arise from. The term meningeal fibroblastoma used by Penfield, however, seems to be the best, as it suggests not only the histogenesis, but also the fact that the tumours may arise from any part of the meninges.

Pathology.

These tumours are usually firm, well encapsulated and elastic, reddish brown in colour on the exterior, sometimes smooth, but in certain sites nodular like a mulberry. They present themselves in two types: the common—spherical with a small area of dural attachment—may reach a very great size; the rare—meningioma *en plaque*—occurs as a flat growth spreading over a large area of *dura mater*.

Being of mesodermal origin, it is not surprising that the tumour does not invade the nervous tissues, but simply pushes the brain with the covering *pia mater* in front of it. Sometimes the tumour makes a nest for itself and may become almost encased in the cortex. The blood supply is almost entirely from the dural vessels or from the superficial cerebral arteries, there being few vessels of any size running from the *pia mater*. These tumours, owing to their slow growth and non-destructive character, may, if they infringe on a silent area, grow to an enormous size and cause surprising dislocation effects on the falx or tentorium. From their habit of growth they are, of course, particularly favourable for surgical attack, and many dramatic cures have been effected. On section, both types vary greatly as regards cellularity and vascularity. While some are hard, others are very soft and vascular. There is, however, little or no tendency towards degeneration or gross cystic change, although in the more fibrous varieties patchy calcareous change and the formation of psammomatous bodies, which give a gritty sensation to the knife, are common.

Involvement of or changes in the overlying bone are well known and seem to be more frequent in the flat tumours. Much controversy has taken place in repect to this interesting phenomenon of an essentially benign tumour infiltrating overlying structures and so exhibiting malignant habits, a finding not paralleled in any other growth. The degree of bone involvement varies from a small degree of pressure atrophy to the production of a large bony hyperostosis. True bony thickening occurs in about 20% of the cases and may occur anywhere in the skull. In the flat type, a large bony thickening is formed by outward spread, the pericranium being often broken through with infiltration of the muscle or overlying soft tissues, so that recurrence is not uncommon after operation. This sort of development is rare in the large spherical tumours, although they do sometimes cause erosion of the bone.

The tumour cells actually invade the Haversian canals and venous channels of the bone, with destruction of the normal bony architecture, which is followed by stimulation of the osteogenic elements and the reformation of an irregular type of bone. In this the bony spicules may be laid down in a radiating manner, very suggestive of the appearance sometimes seen in osteogenic sarcoma of the long bones. Not infrequently we find these two processes of destruction and regeneration of bone going on side by side. Strangely enough, after some time the tumour appears on the outer aspect of the bone, where it may form a soft tumour pad.

In rare cases a typical meningioma may be found completely embedded in the brain without any apparent connexion with the arachnoid or *dura mater*. These cases are very rare, but it would seem that the tumour must then be derived from misplaced arachnoidal rests which have been carried

into the depths of one of the sulci by the pial ingrowth. Microscopically the tumours show a surprising variability as regards their vascularity and cellularity, but the typical cell is similar to that found in the Pacchionian granulations. The nuclei are large and rounded, with a vigorous chromatin network, the cytoplasm being voluminous and granular. The cell boundaries may be indistinct and they tend to arrange themselves in whorls or columns around a collagen fibril or a small vessel. In the more fibrotic varieties the centre of these whorls may undergo degeneration, with the formation of *corpora amylacea*, forming the so-called psammoma. This is often associated with the formation of interstitial collagen, which can readily be detected by special staining methods. The fibrotic type is most commonly seen in the basal regions and in the spinal meninges.

In the more rapidly growing tumours the cells are arranged in columns or quite irregularly, with much less differentiation and more variation in size; they often show mitotic figures. Whorl formation in these tumours is rare, but fibroglia and collagen may be produced. Some of them are extremely vascular and have been classified as angioblastic. Great care has to be exercised in the interpretation of these very cellular and sometimes active growths, while the same care must be taken with recurrent tumours in which the cellular appearance is often modified. Some of these have been classified as malignant meningiomata; but it is probable that they are really sarcoma of the *dura mater*. These sarcomatous tumours, unlike the typical benign growths, tend to cause actual erosion of the overlying bone. Others have been confused with a very similar hæmangioblastoma—a tumour of vascular origin which not infrequently becomes adherent to the *dura mater*.

These tumours are essentially benign in their behaviour, although their local infiltrative effect in the skull and the occasional occurrence of a rapidly growing cellular tumour, which recurs rapidly even after apparently complete removal, gives them a peculiar place in oncology. Rarely, too, they invade the venous sinuses. This may be the source of distant metastases (as in the case with pulmonary metastases recorded by Stewart), and sometimes the tumour may actually grow in the venous stream (as in the case recorded by Towne).

Distribution.

From a careful study of cases at operation and autopsy it is found that these tumours occur most commonly in the region of the great venous sinuses and so are situated in the areas in which the arachnoidal tufts are normally most numerous. It is interesting that they occur much more commonly in the anterior portion of the hemipsheres and in the basal regions of the skull, so that the frontal lobes are more commonly affected than the others.

They are also well known over the distribution of the middle meningeal vessels and are common in the middle but very rare in the posterior fossa. A few cases have been recorded in which multiple meningiomata were present.

They are, of course, relatively common in the spinal meninges, particularly in the dorsal and lateral aspects, and here they comprise the only common benign tumours. Several classical types of tumour with fairly clear-cut clinical syndromes are now recognized. They are the parasagittal tumours, the suprasellar, the olfactory groove, the sphenoidal ridge, the occipital, and those of the convexity and of the spinal meninges.

General Clinical Considerations.

Age.—Meningiomata are tumours of adult life, reaching their peak incidence between the ages of thirty-five and forty-five, so that they are rare, but not unknown, in youth and old age. They comprise about 12% to 14% of all intracranial tumours.

History.—There is usually a history over a period of years, owing to the slow growth of the lesion and to the amazing compensatory power of brain tissue, unless the tumour affects a non-silent area of the brain, when a small tumour may produce symptoms relatively early. In the spinal tumours, too, symptoms will be produced while they are yet small. There seems to be a direct relationship to trauma, so that although trauma is common, there have been very many cases recorded in which the relationship seems to be too close for mere coincidence. It would seem that the active hypertrophy of the arachnoid tufts following the stimulation of injury must sometimes remain as a permanency and initiate the neoplastic process. Cushing, Penfield and Spiller have all commented upon this relationship.

In certain silent areas of the brain the tumour may reach an enormous size, perhaps larger than any other type of intracranial tumour, before it produces any subjective or objective symptoms sufficient to arouse suspicion; and when the tumour is discovered its size often seems to be out of all proportion to the symptoms produced. This latency is one of the most surprising features and is, of course, due to the slow growth and the non-infiltrative nature of such tumours. Later, of course, they may cause distortion of the ventricular system and so internal hydrocephalus and general increase of intracranial pressure.

The great size of many of these growths leads to chronic venous stasis of the region through interference with the normal venous channels, the development of new channels and increased vascular supply to the tumour. These signs may all be manifested in radiographs of the skull, although great care has always to be taken in the interpretation of vascular markings in the skull.

Radiography may be of extreme value in the diagnosis by demonstrating bony erosion or hyperostosis (which may, of course, also be observed clinically) or the presence of calcification within the tumour itself.

It is obvious that other clinical findings will depend almost entirely on the site of the tumour, and I propose to deal shortly with a few classical types.

The Parasagittal Tumours.

The parasagittal tumours[18] classically arise from the arachnoid villi along the anterior half of the sagittal sinus and are common, comprising one-fourth or, with those of the convexity, three-fifths of all meningeal tumours. They are adherent to the *dura mater* near the sinus, but may grow out into the hemisphere, between the hemispheres, or even across the mid-line. Luckily the greater number of them are in front of the Rolandic vein. Their blood supply is almost entirely from the anterior and posterior cerebral arteries.

Clinically there is a long history of some years of unilateral convulsions, often starting in the leg and then involving the arm, sometimes with mild residual paresis of the leg. General pressure phenomena are often very greatly delayed. In the case of those tumours situated in the more silent areas, mental changes, which are often only vague, are almost the only symptoms, so that these patients are sometimes admitted to asylums for the insane. Some cases are particularly hard to differentiate from vascular degeneration without recourse to ventriculography, although the occurrence of bony changes or increase of vascular channels in the radiograph may help in the diagnosis.

Suprasellar Meningiomata.

Suprasellar meningiomata[19] arise from the sinuses around the pituitary diaphragm, occur in late middle age as a rule, are of very slow growth, and produce recognizable symptoms while relatively small, although, owing to non-recognition of the clinical syndrome, they may be allowed to become very large.

They produce primary optic atrophy from direct pressure; thus the patient usually complains of poor vision in one eye before the other, because, owing to one of the nerves as a rule being more affected than the other, symmetrical loss of vision is not common. This early involvement of the optic chiasm makes this tumour of particular interest to ophthalmologists. As a rule the fields of vision show bitemporal hemianopia, more advanced in one eye than in the other. Not infrequently one eye is already blind when the patient comes for examination.

As the tumour enlarges it tends to extend backward, pushing the chiasm backward and upwards, and finally produces a certain amount of dyspituitarism and internal hydrocephalus, with general rise of intracranial pressure because of distortion of the third ventricle, the *sella turcica* remaining essentially normal until very late.

Olfactory Groove Meningiomata.

Olfactory groove meningiomata[20] arise from the anterior fossa meninges of the cribriform plate and extend upwards beneath the frontal lobe and backwards to the optic chiasm and hypothalamic region.

The patients are elderly as a rule, and the first complaint is of failing vision in one eye, followed by mental deterioration. Examination reveals a very characteristic syndrome—that of an elderly person with unilateral anosmia, which later becomes bilateral and complete; unilateral optic atrophy; and papillœdema, with perhaps beginning optic atrophy of the side opposite the lesion, combined with mental symptoms of the frontal type, with at first little, if any, sellar erosion.

As these tumours increase in size the increase of intracranial tension produces headache, papillœdema and secondary atrophy of the sellar region. Owing to the fact that these patients' anosmia is ultimately recognized, fruitless nasal operations have been performed on most of them. Radiography sometimes reveals a small patchy calcification just above the *crista galli*.

The condition has to be differentiated from suprasellar meningioma and other lesions about the optic chiasm. Cushing has reported some very successful cases, the operation on one of which took the record time of seven hours and required two transfusions, but the patient was completely cured. The use of the electric scalpel has revolutionized the operative management of cases like these.

Meningiomata of the Sphenoidal Ridge.

Meningiomata of the sphenoidal ridge[21] grow from the region of the wings of the sphenoid and may be actually parasellar, when they produce signs and symptoms very similar to those of the truly suprasellar growths. Others project back into the middle fossa and invade its floor, sometimes with pressure on the Gasserian ganglion or the temporal lobe. Still others invade the bone of the temporal fossa or the orbital cavity and may produce external deformity in the form of bulging of the temporal fossa, with or without unilateral exophthalmos or proptosis.

It is obvious that if the tumour approaches the mid-line, signs of pressure on the optic, olfactory and oculomotor nerves can occur, with characteristic signs. In addition, unilateral erosion of the sellar region may be shown by radiography, which also often reveals thickening or erosion of the sphenoidal ridge.

Later signs are those due to pressure on the overlying frontal or temporal lobes and to increased intracranial pressure from pressure posteriorly with the production of papillœdema.

Treatment of Meningiomata.

It is a great advantage in these cases to know what to expect at operation—forewarned is forearmed—because what may have looked like a simple procedure may become a procedure of the utmost gravity. Unless a blood donor is ready, and muscle, and an adequate diathermy machine, tragedy may easily occur. The surgeon who attempts to take one of these tumours in his stride, as it were, is riding for a fall.

As a rule, the taking of a detailed history, with or without the use of ventriculography, leads to a correct diagnosis; and although the actual removal of the tumour may be difficult—in fact it may try even an experienced surgeon's skill to the utmost—this is the type of intracranial tumour which is most often amenable to complete removal and cure.

The exposure should be adequate by the turning down of a bone flap, which should include any bony hyperostosis present. This may be a very bloody performance, because, owing to the long-standing stasis, it is usual to find not only the bone but the scalp extremely vascular; so vascular in fact that the operation may have to be abandoned or further attack postponed till a later date, even by an experienced surgeon. It is therefore essential to have at hand plenty of muscle, which can be obtained from other patients and stored in the refrigerator for three to four days, and a blood donor.

The use of electro-surgery has revolutionized our technique in the management of these cases, and a suitable diathermy machine with both coagulating and cutting current must be ready. In all cases adequate exposure, even if part of the overlying cortex has to be excised, is absolutely essential.

The management of the blood vessels is important, and an attempt should be made to obtain control of as many of them as possible, by clips, by ligature or by electric coagulation, before the tumour itself is attacked. It is very tempting to put the finger in and try to enucleate the tumour, but such a procedure has many times been proved wrong, owing to the unnecessary trauma and hæmorrhage, and is very likely to be fatal. The attachment to the *dura mater* must, of course, be removed with the tumour, and during this procedure a certain amount of control of the vessels is obtained. Once exposed, the central portion of the tumour should be looped out with the diathermy loop; this enables the tumour shell to be collapsed or folded so as to make the bed and the small vessels there accessible to clips *et cetera*. By means of packing in strips of lintene between the tumour and the brain substance, the former can gradually be separated and finally removed—a time-consuming but essentially safe method. The sudden removal (in one stage) of a large meningioma may be productive of serious symptoms and even death, in spite of transfusion and other measures. It involves a real risk, and some operators invariably remove large tumours of this type in several stages. The fatal result is apparently due to the sudden decompression and dislocation effects and to localized cerebral œdema following the relief from what has been very long-standing pressure.

The tumours of the convexity are comparatively easy to attack, but those of the parasagittal region may be extremely difficult, and sometimes necessitate removal of part of the sagittal sinus, although the greatest care must be taken not to remove any of this behind the Rolandic vein, as varying degrees of paralysis of the limbs, or even death, will result. It is obvious that in bilateral cases of tumours arising from the falx it may be impossible to remove the area of attachment completely.

In tumours of the base, that is, in those of the suprasellar, the olfactory groove and the sphenoidal ridge regions, which are relatively inaccessible, the brain must be elevated or excised to give access, while the greatest care must be taken to avoid injury to vital areas, such as the infundibulum, the main arteries, or to the optic chiasm. Each of these has its own special technical problems, but in all the use of the electric loop with piecemeal removal and gradual control of the blood supply is essential to success. A great number of dramatically successful cases have now been recorded by a number of surgeons, particularly in America, and the time is coming when, by accurate localization and improved technique, success will be much more common.

This short consideration of a single type of tumour (and as much, if not more, could be written about practically any other type) will indicate that much progress has been made in this new specialty; but we are still only on the threshold of the subject. For its fuller development, groups of special workers must arise, thoroughly trained not only in general medicine and surgery, but also in neurology and in the peculiar tedious operative technique which has been proved necessary. Our present position has been reached only by correlating the work of a great number of observers and discoverers in almost every branch of science, and this correlation must continue if we are to advance still further in the management of these by no means uncommon neoplasms.

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SOME DISTURBANCES OF THE CIRCULATORY SYSTEM DUE TO PNEUMONIA AND OTHER TOXIC CONDITIONS.[1]

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WHEN our Honorary Secretary suggested that I might prepare a paper dealing with some branch of cardiology, I found difficulty in selecting a suitable subject owing to the fact that most common conditions had been well described in the current medical literature.

Rather than bore you with some abstruse problem, however, I prefer to run the risk of doing so with one which is quite commonplace, but which, I believe, has not been clearly visualized by all practitioners. I refer to the toxic effects on the circulatory system of such diseases as pneumonia, influenza, typhoid and typhus fevers, hyperthyreoidism *et cetera*. I am well aware that some would exclude the thyreoid from this discussion, but there are those who hold that there is a toxic element in certain cases, whence the term dysthyreoidism. In view of the present state of our knowledge, their opinion must still command our respect, even if not our acquiescence.

I propose to use pneumonia as an example before drawing comparisons by means of brief summaries and proffering certain deductions and generalizations.

The Manner in which the Circulatory System is Damaged.

The prime factor is the toxin, which may poison both the heart itself and the peripheral vascular field, but of the two the latter effect is probably the more important. Even in the absence of myocardial damage serious injury may be inflicted on the capillaries and arterioles. One must also consider the mechanical factors in the causation of failure of the heart.

They include the following: (i) The capillary stasis in the diseased lung tissue diminishes the oxygenating area, producing temporary anoxæmia, which in turn increases the output and work of the heart. (ii) Interference with respiration hampers diastolic filling. (iii) The fever causes an accelerated basal metabolic rate and so increases the heart's work. (iv) Cough increases the work and interferes with diastolic filling. (v) Emotional disturbance increases the work of the heart.

Important as these factors may be, the normal heart has sufficient reserve to cope with the extra strain, but if the myocardium is damaged by the toxin or hampered by preexistent cardio-vascular disease, then the mechanical factor may be the last straw precipitating cardiac failure.

Peripheral failure, to which I most particularly call your attention, is a condition akin to shock. In it the walls of the small peripheral vessels are so damaged by the toxin that they lose their tone and dilate. The result is that the blood accumulates in them until, in fatal cases, the venous return to the heart becomes inadequate for the maintenance of the circulation. In the majority of patients the damage is not so severe, but produces signs in proportion to its degree.

Lesions Found in the Heart.

Usually there is nothing more in the heart than some cloudy swelling of the fibres, rendering the striations indistinct. Frank myocarditis is rarely present, although degenerative or hæmorrhagic lesions are sometimes found *post mortem*; but this occurs mainly in persons dying toxic deaths from the disease itself.

If true cardiac failure has occurred, the right side of the heart will be found to be dilated, for the main strain falls here. Endocarditis and pericarditis have been seen, but there are no cardiac lesions that are typical of this disease.

The pathological lesions are generally ill-defined; and one must remember that the heart may have functional disturbance without obvious lesions.

Summary.—In diphtheria the changes may be the same as those described for pneumonia, but reports are contradictory. Hæmorrhagic lesions and perivascular leucocytic infiltration may be present. In one series of one hundred cases a parallel was found between the degree of the organic lesion and the change in the *P-R* interval.

In typhoid fever the lesions are very slight.

In typhus fever the lesions are very pronounced. They are on a par with those of acute rheumatism, and consist of acute inflammatory lesions, interstitial infiltration and infarcts from thromboarteritis. Apart from some fibrosis all the lesions clear up.

In hyperthyreoidism no characteristic lesions are found. An increase in the weight of the heart up to 15% may be found. There are increased diameter and pulsation of the pulmonary artery. Occasionally pericardial thickening may be found. Some observers hold that no damage is due to the thyreoid alone, but Parkinson does not agree with this view for all cases.

Signs of Heart Failure.

Signs of heart failure are usually not prominent, owing to the rapidity with which the patient dies

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 26, 1936.

from the effects on the central nervous system of a toxæmia sufficiently severe to cause myocardial failure. Of course, this does not obtain in the presence of preexistent cardio-vascular disease, in which even mild toxæmia may precipitate failure. When death occurs from myocardial failure, it is preceded by a more or less rapid dilatation of the right ventricle, with enlargement of the liver and engorgement of the veins in the neck.

Elevation of the venous pressure in pneumonia probably occurs only in the presence of heart failure,[18] but the evidence is somewhat contradictory.[7]

Auricular fibrillation is of rare occurrence, Cohn and Lewis[5] finding only 18 examples among 1,456 cases, and then usually in association with fatal heart failure. It is to be noted, however, that in 10 of their cases normal rhythm was resumed prior to death. In some young patients it seemed possible that digitalis precipitated this abnormal rhythm.

The electrocardiogram may show an alteration in the *T* waves, but in pneumonia, unlike diphtheria, heart block is unknown, except as a result of overdosage with digitalis.

Summary.—In pneumonia electrocardiographic changes are inconstant; auricular fibrillation is rare.

In diphtheria changes, especially in the *P-R* interval, run parallel with danger. Eleven out of seventeen patients with heart block died.

In typhoid fever the electrocardiographic changes are unimportant.

In typhus fever 80 out of 85 patients had clear-cut changes in *P*, *T* and *R* waves and in the *P-R* interval between the ninth and forty-fourth day. Three had auricular fibrillation. In 85% anomalies of *Q*, *R*, *S*, *T*, and in 48% altered *R* and *S* waves were found. The changes are due to the severity of the attack. Recovery, if it occurs, is absolute.

In hyperthyreoidism 25% of patients have an altered *T* or *P* wave. The axis shifts to the left. There is no great prognostic value in the changes. Auricular fibrillation is observed in from 6% to 27% of cases. The change is functional, because 60% to 75% return to a normal rhythm after operation.

Summary.—In more than 50% of cases of pneumonia, Levy[11] by X ray examination found a gradual increase in the size of the heart, the maximum size being attained at the height of the disease.

In diphtheria, typhoid fever and typhus fever radiological changes are rarely observed.

In hyperthyreoidism an increase in size was observed in 26% to 50% of cases in different series. A great increase in size occurred only with auricular fibrillation or congestive failure. An increase in the pulsation and diameter of the pulmonary artery may precede enlargement in 33% to 54% of cases. The superior *vena cava* throws a prominent shadow, and the aortic arch may be raised. The heart is ham-shaped.

Peripheral Failure.

In the *post mortem* examination of 200 cases of pneumonia Brooks[3] found that peripheral failure had occurred in 109, this number including all the 77 patients who died from cardiac failure. In these there were dilatation and congestion of the capillaries and veins.

The condition may be compared with shock, in which a histamine-like substance is liberated to cause dilatation of the peripheral arterioles and capillaries with increased permeability of their walls. Blood stagnates here, causing inadequate venous return to the heart and eventually death from lack of blood supply to the vital centres. In the more toxic forms of pneumonia the bacterial toxins may lead to a slow liberation of the histamine-like substance from the cells over a prolonged period. This effect on the peripheral circulation was first suggested by Brodie[2] in 1899 in relation to diphtheria, and then proved experimentally by him. That dilatation of the terminal vessels does occur owing to the toxins poisoning the walls can be demonstrated[15] by the skin response to adrenaline, which shows a diminished increase in resistance in the vessels which normally contract as a result of the injection.

Recovery of tone in the vessels affected by the toxæmia may be very slow and not complete until long after the crisis.

It is said that the damage to the walls of the arterioles is in the neuro-muscular connexions, so that loss of control of the arterioles by the splanchnics occurs. (This is probably more noticeable in diphtheria.)

The vasomotor centre has been shown to be intact.

Signs of Peripheral Failure.

The two main signs by which peripheral failure may be recognized are a falling blood pressure and cyanosis.

The systolic pressure often falls slightly in the early stages of pneumonia, but a great fall is usually due to peripheral failure leading to insufficient filling of the heart. In these circumstances the pressure may fall below 100 millimetres of mercury in a young adult, or to a corresponding level in older people, this being of grave prognostic significance. In consequence the pulse is small, thready and rapid until it gradually disappears.

Cyanosis is produced by the dilatation of the capillaries and a decreased amount of blood flowing through them in unit time, while in some cases anoxæmia also is a factor. The slowing of the blood stream is evident when the blood returns tardily to a patch of skin which has been blanched by pressure.

Cyanosis is not a prominent feature in lobar pneumonia; in fact Murray Leslie[13] states that in 90% of all cases no cyanosis ever occurs. It is in the more toxic cases that we find it, and also in bronchopneumonia when there is the added factor of anoxæmia.

In the virulent influenzal pneumonia of 1918 the heliotrope colour was a striking feature, and in spite of the absence of any signs of cardiac embarrassment it was soon recognized as the prelude to a fatal ending. This cyanosis was largely the result of slowing of the peripheral circulation due to the action of the toxin on the terminal vessels.

In acute toxæmia other symptoms of peripheral failure may be produced by many lesions and so are not of diagnostic value; but during convalescence they are the cause of much discomfort. The patient is troubled by sensations of feebleness, faintness, postural giddiness, breathlessness on slight exertion, palpitation *et cetera*. These are in the main due to a loss of control of the distribution of the blood.

As the peripheral vessels gradually regain their tone the blood pressure rises, the pulse slows and improves in volume, the response to effort improves, and the symptoms slowly disappear.

Summary.—In diphtheria peripheral failure is very important. There is a latent period and the patients often die in the first week. In the pallor it differs from pneumonia.

In typhoid fever peripheral failure may occur.

In typhus fever peripheral failure occurs early. It is most pronounced on the tenth day. The blood pressure may be as low as 80 to 50 millimetres of mercury. The hypotension slowly disappears in convalescence.

Generally, when recovery occurs, it is slow but complete.

Myocardial failure occurs sometimes in pneumonia; it occurs early if the toxæmia is very severe.

In diphtheria myocardial failure is recognized as a cause of death; it may occur in the second week or later, or even two weeks after the fever has gone.

In typhoid fever myocardial failure is extremely rare.

In typhus fever it is common. It may occur early, but it occurs most often on the tenth day. Thirteen of eighty-five patients died in the first fortnight. Peripheral failure is usually present as well.

In hyperthyreoidism we find that Parry's first patient died of heart failure in 1815. Heart failure occurred in 39% of patients over forty and in 7% of those under forty years of age. The causative factors are: (i) old cardio-vascular disease, (ii) overwork from a high basal metabolic rate, (iii) auricular fibrillation. Diagnosis is very important because myocardial failure is an urgent indication for operation. Generally preexisting cardio-vascular disease is a very important consideration.

Prognosis.

What Hay[9] says regarding lobar pneumonia is in effect that 70% of patients recover whatever the treatment, 15% die in spite of treatment, 15% recover if treated adequately. From this it is seen that in 15% the toxæmia may be so severe as to render our methods of treatment futile.

Brooks[3] found in his 200 *post mortem* examinations that whereas 73 cases exhibited acute cardiac lesions, no fewer than 116 patients had had chronic cardiac disease.

These figures provide striking evidence of the danger of infections such as pneumonia to people suffering from cardio-vascular disease, and also tend to show why the prognosis in pneumonia is so much more grave in the elderly.

Unfavourable signs include: (*a*) a pulse rate of more than 130 per minute; (*b*) a low systolic blood pressure, for example, under 100 millimetres of mercury in a young adult, or a little higher in an older person; (*c*) great cyanosis; (*d*) dilatation of the right heart with venous congestion.

The ultimate prognosis in patients who survive in spite of myocardial damage is good, unless, of course, they have a chronic cardio-vascular trouble which has been aggravated by the acute toxæmia.

Bedford,[1] referring to influenza *et cetera*, writes: ". . . available evidence suggests that they do not cause permanent myocardial injury."

Those who survive peripheral failure usually make a complete recovery, but the process may be extended over a period of months.

Summary.—In diphtheria there is danger proportional to electrocardiographic change; if the change is very abnormal, the prognosis is grave. Patients rarely recover if the systolic blood pressure is under 65 millimetres of mercury. Vomiting with no obvious cause is grave, often being due to a cardiac reflex. Patients die in three days, maybe in twenty-four hours, from the onset of failure. Peripheral failure may cause sudden death in the first week. Probably no permanent change takes place in those who recover.

In typhoid fever there is no trouble.

In typhus fever there is an alteration in the *T* wave of the electrocardiogram proportional to the severity. Sixteen of eighty patients died.

In hyperthyreoidism the main risks are: (i) previous cardio-vascular disease, (ii) auricular fibrillation, (iii) senile changes in the heart. The operative mortality is only about 1%; the operative mortality in failure is 4% to 20%, emphasizing necessity for early operation and cooperation with the surgeon. Permanent damage, if any, depends mainly on previous cardio-vascular disease.

Treatment.

Treatment of Heart Failure.

If venous congestion occurs, or if the right heart dilates, venesection to the extent of not less than twelve ounces is indicated.

If there is much anoxæmia, oxygen should be given continuously, day and night, at the rate of two litres per minute through a nasal catheter in the absence of a tent, or by subcutaneous injection, for in some cases anoxæmia is a factor in precipitating cardiac failure.

The routine administration of digitalis is advocated by some and deprecated by others. Before it is considered, the physician must remember that peripheral failure is more common than myocardial failure. Therefore it is in only a limited number of cases that there is any scope for its beneficial action. In the remainder it is tantamount to adding yet another poison to a system already beset by toxins the effect of which on the heart may be aggravated by the action of the drug in constricting the coronary arteries. In one series of cases it appeared that digitalis might have caused auricular fibrillation in a few young patients.[5]

Statisticians can prove anything, but when rival groups obtain results that negative one another, we cannot do otherwise than defer judgement.

Stone[17] found in the records he investigated that the death rate was lower among those receiving digitalis than among the remainder; but Burrage and White[4] showed that the cases were not suitably chosen and therefore the results were not conclusive. Their results, also subject to criticism, showed that the mortality with small doses was three times that with full doses, while Niles and Wyckoff,[14] who exhibited digitalis to alternate patients, found the mortality greater in those who received massive doses than in those who received none.

In considering these results, it must be remembered that it is very difficult to estimate the dose necessary in the presence of normal rhythm. As a large dose is probably necessary, the obvious risk is due to the fact that the margin between saturation and intoxication is so small. When auricular fibrillation is present some have found digitalis of definite value, while others have been unimpressed.

Until we have proof that the routine administration of this drug is of value I do not think it advisable to give it indiscriminately, but, if it is to be given at all, would suggest reserving it for those cases in which there are definite indications, such as myocardial failure.

As massive doses are risky and smaller doses are rather slow in exerting their effect, while in the presence of cardiac failure the need for digitalization is urgent, the use of strophanthin E, or strophanthin G ("Ouabaine"), intravenously is to be recommended. Here, by giving 0·43 milligramme (one one-hundred-and-fiftieth of a grain), a full dose, the effect is obtained in fifteen minutes. In order to avoid the risk of toxic effects it is usually wiser to administer several fractional doses, when the result may still be obtained in six to eight hours. Digoxin may be used in a similar manner.

Rest, obtained by adequate doses of morphine if necessary, is an essential part of treatment.

Treatment of Peripheral Failure.

Before discussing the treatment of peripheral failure I wish to focus your attention on the condition that obtains in the vessels.

The small arterioles are so damaged by the toxins that their muscular walls have lost tone and no longer respond adequately to the nervous stimuli which normally control the distribution of the blood. In addition, the walls of the capillaries and terminal arterioles have been damaged, resulting in loss of tone and dilatation, so that an excess of blood is accumulated there.

In pneumonia, even apart from peripheral failure, the skin capillaries contain a great deal of blood, probably for the purpose of discharging some of the heat from the febrile patient.

It is easy to see why hydrotherapy meets with some measure of success in cases of peripheral failure, because it reduces the temperature and at the same time constricts the terminal vessels, so helping the peripheral circulation. Camphor appears to withdraw blood from the skin, but its tendency to dilate the heart does not enhance its popularity.

Alcohol is distinctly contraindicated, for among its ill-effects is dilatation of the peripheral vessels, which is absolutely adding insult to injury. Our sheet anchor is adrenaline, for it not only causes constriction of the small arterioles by acting on their muscular walls, so raising the falling blood pressure, but, what is more, it increases the force of the heart's contraction and dilates the coronary vessels.

These beneficial attributes are undoubtedly very valuable in mild cases, but, when the toxic damage to the walls is sufficiently severe, even adrenaline fails us, and we then have no drug that will prevent the fatal issue. The reason for this is that the peripheral vascular paralysis is brought about by a substance similar to histamine, which Lewis[12] showed to exert a far stronger local action than adrenaline, so causing a passive dilatation of the vessels. However, in the presence of peripheral failure it is well worth while trying subcutaneous injections of adrenaline, 0·3 cubic centimetre (five minims) every two hours, always remembering, however, that some people are very susceptible to this drug, which acts as a cardiac poison if even a slight overdose is given.

The second string to our medicinal bow is pituitrin, which Krogh[10] suggests may be the substance normally controlling the size of the capillaries. It may be given in addition to adrenaline in doses of one cubic centimetre subcutaneously every twelve hours, because its action is more prolonged.

However, apart from its action in constricting peripheral vessels and so raising the blood pressure, it unfortunately tends to dilate the heart and to constrict the coronary vessels. Therefore it behoves us to make certain that the myocardium is practically unimpaired before throwing on it the extra strain of increased blood pressure, dilatation and diminished coronary supply, although it must be remembered that an increased blood pressure necessarily increases the coronary flow.

In toxic cases this is obviously difficult to determine, and careful must be the man who wields such a two-edged sword. Mention must be made of strychnine, which acts as a respiratory and vasomotor centre stimulant. If the toxic effect on the walls of the vessels is present, reflex contraction is not likely to be obtained. It is in convalescence that this drug is most likely to help. Intravenous injections of 30% to 50% solutions of glucose help.

Treatment in Convalescence Applicable to Either Type of Failure.

During convalescence the sense of weakness, combined with the inability to undertake exertion without provoking palpitation and breathlessness, is apt to have a very depressing effect on the patient. Frequently he imagines that his heart is seriously damaged. It is the duty of the physician to reassure and encourage him and by gradual physical training to convince him that he is steadily improving.

While still confined to his bed, he should receive massage and exercise,[6] the former directly assisting the venous return, while the latter, by improving the tone of the muscles, indirectly aids this process. When expense of a masseur precludes his attention, breathing and other mild progressive exercises must be prescribed, for example, first the use of the bedpan may be abandoned, then the patient may wash himself and do arm and leg movements.

Of course the pulse rate will increase during this treatment, but the masseur should apply soothing vibratory massage over the chest at the end of each treatment to insure that the final rate is not higher than at the commencement. When the pulse rate has not exceeded 80 per minute for a period of three days, it is time to get the patient out of bed, but under close observation lest the rate rise unduly.

Subsequently the exercises are gradually increased; none, however, are allowed that cause an increase in heart rate which does not return to normal after two minutes' rest. By this means not only does the improved muscle tone assist the circulation, but the heart muscle itself is improved. These methods were introduced by Schott on the Continent, but they have since been scorned by some. After careful investigation of a series of cases, Proger and Korth[16] made one discovery, namely, that patients with combined mitral stenosis and aortic regurgitation showed a very definite improvement after training, while those with mitral stenosis alone did not improve. They ascribe the improvement to increased cardiac efficiency, improved vital capacity, and enhanced neuromuscular efficiency by practice.

That this last suggestion is feasible is supported by the well-known fact that many patients are able to do hard work to which they are accustomed, but are unable to cope with an unfamiliar task entailing much less physical exertion.

Correctly used, the Schott method amply justifies itself in the "after treatment" of all toxic disturbances of the circulation, and more especially when peripheral failure is the predominant feature.

Summary.

1. The toxins of pneumonia *et cetera* may cause myocardial failure, peripheral failure, or both.

2. Excepting in typhus fever, frank myocarditis is comparatively uncommon in this group.

3. Emphasis has been laid on the grave danger in chronic cardio-vascular disease of an infection which may precipitate cardiac failure.

4. Evidence points to the fact that normal hearts rarely receive any permanent damage from the toxins under consideration.

5. The most important factor in producing permanent damage, even in hyperthyreoidism, is pre-existing cardio-vascular disease.

6. Digitalis is of doubtful value in most cases, even where auricular fibrillation is present; it is suggested by some that it constitutes an additional risk.

7. Peripheral failure has been shown to be an important feature of very toxic cases and may be the cause of death, especially in diphtheria. It is the cause of most of the symptoms during convalescence in this group.

8. Treatment, including the administration of adrenaline and pituitrin, has been described, but from the very nature of the pathological process it is evident that when the toxæmia is very severe there is no known treatment that will avert a fatal termination.

9. To conclude in brighter vein, it is pleasing to be able to assure the convalescent patient that he is on the road to certain and complete recovery.

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ON THE SEQUELÆ OF CEREBRAL INJURIES DUE TO EXTERNAL TRAUMA.

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IT will be freely admitted that our understanding of the pathological anatomy of brain injury is at present totally inadequate and uncertain. Our ignorance of the processes underlying the production of the many and varied sequelæ of *commotio cerebri* is even more abysmal.

The rapid increase of speed in industry and transportation has led to a corresponding increase in severe injuries of all types, of which head injuries form no small proportion. I am unable to obtain statistics for Australia, but G. W. Swift states that each year in America head and spine injuries number approximately 250,000.

Those of us in hospital or general practice are all familiar with the more or less immediate sequelæ of head injury. I refer to concussion; coma, with or without a lucid interval; localizing neurological signs, such as unilateral mydriasis and hemiparesis; and all the various clinical signs indicative of intracranial hæmorrhage.

It would be impossible, without writing a textbook, to do justice to such a vast clinical field. It is rather my purpose to draw attention to the importance of the more prolonged sequelæ of cerebral trauma, as I am of the opinion that, in the average practice of medicine and surgery, they do not receive the care and clinical study which they deserve.

Consider the case of a week-end motor speedster admitted to hospital after a road smash and suffering from injuries about the head. He is put to bed and his clinical symptoms and signs are met with the routine treatment for such cases. The honorary surgeon may or may not see him. The resident medical officer, if he is careful, will have an X ray picture taken of the skull. If no fracture is detected and the patient appears comfortable, he is allowed to rest for a few days and is then discharged. A follow-up system is seldom, if ever, employed and very often no advice has been given to the patient regarding the possibility of sequelæ affecting him in the next two or three months. Nothing may happen and the patient may keep well. On the other hand, he may find himself later visited by violent headaches; he may develop various reactions due to neurological sclerosis. Infection with subsequent meningitis is a possibility, or at a later date he may develop changes in his disposition, or, less commonly, epilepsy and even disseminated sclerosis. Amnesia and insanity have been known to occur after cerebral trauma. Suicide as a remote sequel is not unknown.

Let us examine these various conditions with more detail.

Headaches.

The headache following cerebral injury is usually due to interference with the mechanism by which the intracranial pressure is regulated, for example, in cerebral œdema. It is of organic cerebral origin and may be accompanied by tinnitus and changes of disposition.

During this week I saw a young man, aged twenty-three years, who sustained a fracture of the cranium twelve years ago and a fracture of the left parietal bone in a motor cycle accident three months ago. He complains of violent bilateral temporal headaches, giddiness on exertion, and lack of ability to concentrate. X ray films show no bone depression in the skull, although the fracture markings are visible. His disposition is such that one is restrained with difficulty from kicking him out of the consulting room. Although well educated, he is loutish and impudent. He even regards the death of his *fiancée*, who was killed while riding pillion on the cycle at the time of his accident, as a lucky affair for himself, because, as he states, "he was getting tired of her anyway".

It is almost unbelievable that this could be the normal disposition of a young, sane man.

Brain lesions due to *contre-coup* will often produce this type of sequel months after the accident.

The end results of changes in a patient's temperament are difficult to follow with statistical accuracy, but Dr. S. J. Minogue, who has closely studied the records of 3,000 suicides in New South Wales, informs me that in seven or eight cases every year a prior medical history of head injury can be obtained. This represents approximately an incidence of one case in forty, a proportion which one cannot disregard as negligible.

Infection.

A potent and dangerous sequel of head injury is the supervention of infection of the meninges. This generally occurs within a few days of the injury and is well illustrated by the following case.

T.C., aged eighteen years, was admitted to the Newcastle Hospital on May 2, 1936, with a history of having been thrown from a motor car onto his head. On admission to hospital he was conscious and had a little cerebral irritation. Ten minutes after admission he had a seizure of the Jacksonian type and blood gushed from his left nostril. He then became unconscious. His pulse rate was 64 and his temperature 36° C. (96·8° F.). He had a lacerated scalp and a depressed fracture of the left temporal bone. One hour afterwards he regained consciousness and, although his speech was incoherent, he understood an instruction to put out his tongue.

His condition improved until May 6, when he complained of severe and increasing headache and of a pain at the back of his neck. He was very restless, his temperature rose to 38·5° C. (101·3° F.), and cerebro-spinal fluid was noticed flowing at times through his left nostril.

Lumbar puncture was performed and twenty cubic centimetres of cerebro-spinal fluid were withdrawn under greatly increased pressure. The fluid was straw-coloured and definitely turbid. Pathological examination revealed it to contain pneumococci.

On the following morning his condition became much worse and he died.

Similar cases of meningitis following fractures of the skull are comparatively common. Other infective complications, such as cerebral abscess and sinus thrombosis, are numbered among the possible sequelæ.

Amnesia.

After head injuries accompanied by immediate loss of consciousness certain disturbances of memory are well known to occur. On full recovery of consciousness the patient is found to have little or no recollection of events during the period of confusion following the injury, nor does he remember the moment of injury. Further, there is sometimes permanent amnesia for a short period before the injury occurred.

Ritchie Russell investigated the duration of this retrograde amnesia in 200 cases at the Edinburgh Royal Infirmary and found that in 180, after full recovery, it proved to cover only a few minutes and in only four cases did it cover more than twenty-four hours, the longest period being seven days. He also investigated the total period of amnesia following the injury and found that in 153 cases it was less than twenty-four hours and in only ten cases did it exceed two weeks. He has never known a permanent retrograde amnesia to cover more than a week prior to the injury, although writers of sensational fiction would have us believe otherwise.

Hysteria, of course, must be eliminated, and it is also interesting to note that the memory for distant events tends to return before the memory of more recent events. This is exemplified by a case reported this week in the Melbourne Press, headed "Mystery Man Doesn't Know Own Name".

He recovered consciousness in the Royal Melbourne Hospital two days after having been found lying unconscious in the street. He could not say what his name was, nor could he believe that he was in Melbourne, although he remembered having visited Australia some years ago. He could describe Bath, England, in detail and says he was born there. He is doubtless suffering from temporary amnesia following a head injury.

In connexion with the subject of post-traumatic amnesias one should mention the automatic states which often follow immediately upon a head injury.

Those who are patrons of the fistic art will perhaps recall having seen a boxer knocked out by a heavy blow on the head and then having later watched him, apparently partially recovered, endeavour to continue to fight wildly, despite the efforts of his seconds to restrain him. In a simpler case the subject is dazed, does not realize where he is, and talks confusedly for a few minutes before coming to himself, as a rule rather suddenly.

Trotter explains this by stating that when concussion has been slight, it sometimes happens that, though consciousness is recovered rapidly, the mental state does not at once return to the normal. Hence the patient is liable at this stage to perform various complicated acts of an automatic character, often absurd or even dangerous. Trotter considers that this "automatism" is of considerable medico-legal importance, for the subject of it cannot be regarded as responsible for his acts.

Traumatic Epilepsy.

Briefly, the causes of epilepsy following head injuries are these:[7] (i) in penetrating injuries, infection of the meninges and brain; (ii) in a small number of cases in which the fits occur within a few hours or days of the injury, epidural, subdural or subarachnoid hæmorrhage; (iii) in the great majority of cases the cause is direct injury to the brain substance occurring at the moment of injury and followed by subsequent cicatricial sclerosis.

A distinction must be made between early fits due to the second group of causes and late fits caused by the third group. The latter form the common type and seldom commence earlier than two or three months after the injury. Of these, 85% take the form of generalized seizures, and the commonest variety is that in which minor are followed by major attacks.

An illustrative case from my own practice is as follows:

W.G., aged eighteen years, was admitted to the Newcastle Hospital on July 19, 1928. His mother stated that three years previous to his admission, while working in Walsh Island shipyard, he fell ten feet down a hold and struck the back of his head on a beam. He recovered from his injury within three weeks, but was left with a "dent" in the back of his head. Prior to his accident he had been a strong, healthy boy.

Three months prior to admission he had commenced taking fits (four altogether—three at home and one at work). His mother described one at home as starting with a cry and going on to quivering and convulsions. He was apparently unconscious and he "wet the bed".

On examination he was a well-developed youth with intelligence slightly below normal. He stated that he did not know when a fit was coming on. At the back of his skull there was a distinct depression running transversely across the occipital region, six centimetres above the external occipital protuberance. It was about three centimetres wide and four centimetres long, and apparently one centimetre deep at its centre.

On his second day in hospital he had a fit, which was seen by the ward sister. She describes it as starting with a loud groan; the patient's face then began to twitch, his teeth clenched and he became cyanosed. Both hands began to twitch with simultaneous clonic movements, his knees were drawn up and he voided urine involuntarily. The attack lasted two minutes. Five minutes after the attack he was able to mutter incoherently in answer to the resident medical officer's questions, his limbs were somewhat spastic, and his corneal reflex had returned.

Two and a half years later he returned to my care with a history of more frequent fits. He was then averaging three to four per month. At the request of his relatives and, it must be confessed, against my better judgement, I performed a decompression operation over the depressed area of the skull. The depressed bone was thicker than normal, and underneath the adherent dura was an area of cicatricial sclerosis in the brain.

Although during recent years surgeons[3] have advocated excision of the cicatrix whenever it can be located, I preferred to have my patient recover and so did not attempt to remove the neurological scar tissue.

The patient recovered well, but returned in three months' time to tell me that he had had a minor fit. He has been examined by consultants both here and in Sydney, but his fits have steadily persisted, until at the present time he presents the typical picture of a chronic epileptic.

Whether or not in this case an area in the forebrain has been affected by a *contre-coup* injury I am unable to say. It is certainly possible.

It is now generally agreed that traumatic epilepsy, once established, tends to persist, that the fits become more frequent and severe until they are a cause of total disability both for the manual and the mental worker. Its frequency following head injuries of all kinds is estimated by Symonds at between 4·5% and 8%. Its frequency after gunshot wounds is from 12% to 15%.

Organic Nervous Disease.

Head injuries in recent years have received attention in work done on the ætiology of organic nervous disease. Wilfred Harris estimates that 7% of 234 cases of disseminated sclerosis originated in head injuries.

Where organic nervous disease already exists, it may be greatly accentuated by concussion or similar cerebral injury. The same author quotes a case in which, in an army officer who gave a positive Wassermann reaction, a fall from a rapidly descending parachute subsequently was followed by the onset of general paralysis of the insane. He was apparently in perfect health until the time of his fall.

Hysteria.

I include this section among the sequelæ of head injury with great diffidence. Undoubtedly it is encountered pure and unadulterated, but the difficulty the practitioner faces is in making his diagnosis untroubled by thoughts that after all he may be doing his patient an injustice and missing a syndrome of organic cerebral origin.

Encephalography is sometimes a help in the diagnosis. Dr. Lyle Buchanan, of Sydney, recently demonstrated three cases of alleged malingering in which the encephalograms showed in one case definite evidence of neuroglial wasting, and in the other two cases evidence of intracranial hæmatomata.

On the other hand, it is an undeniable fact that the victim of a head injury, finding himself a centre of interest for the attention of relatives and friends, sometimes exaggerates his disability. Thus a self-centred patient may pass on easily into that controversial stage known as traumatic neurasthenia. Sir John Collie aptly quotes Duprey as saying:

The psychopathic process generally follows a course which may be summed up in the following words: Commotion, emotional suggestion, exaggeration, simulation, and a claim for compensation.

I relate the following case from my own experience; in this instance the insurance referee diagnosed hysteria, but may have been unfair to the patient in doing so.

S.S., a steel forger, was brought to my surgery on the night of July 13, 1933. He was a tall, well-developed Swede, aged forty-eight years. He stated that he had been struck a glancing blow on the forehead by a piece of steel.

He had a small lacerated wound of the scalp in the middle of his forehead, requiring three stitches, and no injury appeared to have been done to the bone. He did not appear shaken, treated the matter lightly and was able to walk home.

Next morning I was called to see him at his home. He was complaining of violent headache and giddiness and was unable to sit up in bed. There was no doubt in my mind that he was suffering from a severe stage of cerebral reaction. I also saw the "piece of steel" which had hit him. It consisted of the jagged end of a long lever. It was a foot in length, three by three inches in width, and weighed about twenty pounds. When I saw it, I marvelled that the patient was still alive.

He regained his normal health with rest and sedatives, but it was three months before he was able to face his previous job with the steel company. He was uneasy in the job, had no confidence in himself, was called a malingerer by the insurance company, and finally was dismissed by the manager for incompetence.

This man's record prior to the accident was good, and I am convinced that the head injury was the sole cause of his incapacity.

Death.

Death is the oft-times inevitable and final sequel. It occurs, I venture to say, more frequently than it should. Swift, in a series of 214 cases, claims, by improved use of dehydration methods of treatment, to have reduced the mortality to 19·1%. Ten years previously the mortality rate in his hospital was 60%.

Children seem to suffer less from sequelæ than adults. In old age the mortality is influenced by the physical frailty or otherwise of the patient. Cerebral damage in new-born babes, subjects of difficult delivery, is well known, and death is not uncommon four or five days afterwards. At *post mortem* examination subdural hæmorrhage and cerebral bruising are generally found.

I often wonder whether the frequent falls and "bumps" on the head which every toddling child receives between the ages of one and four play any inhibitory part in its subsequent mental development.

I have ascertained from the Newcastle Hospital records (appended) that in the five-year period 1931 to 1935, inclusive, one-third of the total accident admissions were for head injuries. Many of the patients were admitted for short periods of observation from the casualty room; thus we find that the

general death rate is not unduly high—8·7%. Among nine cases showing intracranial hæmorrhage and cerebral irritation, death occurred in seven—a very high percentage.

These figures, of course, do not include patients with minor head injuries treated in the casualty room and allowed to go home, or persons dead on arrival at the hospital, the latter averaging two or three per year.

Except in occasional cases, one or two of which are quoted in the foregoing text, the records of remote sequelæ have not been indexed. Otherwise the record is a fair summary of the head injuries occurring in an industrial and seaport population of 100,000 people.

Newcastle Hospital Accident Admissions, January 1, 1931, to December 31, 1935.

Total number of accidents of all types ..	2,202
Total number of brain and skull injuries ..	764 (34·7%)
Number of deaths from brain and skull injuries	67 (8·7%)
Average days' stay in hospital (brain and skull injuries	9·56
Included in Brain and Skull Injuries:	
Fractured skulls (X-ray examination made)	174 (22·9%)
Intracranial hæmorrhage, cerebral irritation (with 7 deaths)	9
Gunshot wounds (with 1 death)	3
Concussion and lacerations	578
Total	764

Conclusions.

1. In an industrial and seaport community of 100,000 people head injuries form a large proportion (over one-third) of all severe injuries.

2. In brain injury the mortality is relatively high and could be greatly reduced by closer observation and understanding of the clinical phenomena.

3. Sequelæ of head injuries often pass with their true origin undiagnosed.

4. A careful follow-up system of clinical recording is essential in accurately estimating their frequency.

Acknowledgement.

I am indebted to Miss M. Towns, of Newcastle Hospital clerical staff, for her help in preparing statistical information.

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HUMAN PSITTACOSIS IN AUSTRALIA.[1]

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IN the summer of 1934-1935 investigations were made into the incidence of psittacosis in the common species of Australian parrots in the wild state. It was found that a large proportion of recently caught individuals of *Psephotus hæmatonotus, Trichoglossus* (two species) and *Leptolophus hollandicus* were infected, while in other genera examined demonstrable infection was rare, but enlarged non-infective spleens were common. It was recorded[1] that the virus strains derived from wild Australian parrots appeared to resemble closely those isolated from human and parrot infections in Europe and America. As far as we can ascertain, prior to the publication of those investigations, in four instances only had the clinical diagnosis of psittacosis been suspected in Australia, and in none had the clinical suspicion been confirmed by laboratory investigation, though in one case it had been proven that the parrot associated with the patient had died of virulent psittacosis.

It was difficult to account for this apparent absence of human infection in a country where members of the different genera of parrots are frequently closely associated with people as pets. Moreover, the habit of keeping parrots, particularly budgerigars, as pets has increased during the past few years. It was felt that one explanation of the relative absence of human infections reported might be found in the ignorance of the public and the profession of the possibility of human infection: that in Australia human infections of psittacosis might be occurring unrecognized. It was decided, therefore, to publish informative articles in the medical and lay Press and to ask physicians to report any suspicious case to the institute.

Methods of Investigation.

On receipt of a report of a suspicious case, help was given to the attendant physician to confirm or exclude the diagnosis of psittacosis by such means as examination of the blood serum for agglutination against *Bacillus typhosus* or *Brucella*, radiological examination of the chest, and examination of the sputum. In the city, the patient's home was visited in order to obtain a complete history of his parrots from his relatives. A number of his birds were killed and taken to the institute for examination. It was found that the most satisfactory method of dealing with parrots in the homes was rapid drowning in a solution of cyllin. If live birds were sent to the institute, they were dealt with as in previous investigations. The cage was completely surrounded by a cloth soaked in weak lysol and coal gas was run in to kill the bird. After the dust

[1] This work was carried out under a grant for research on virus diseases from the Rockefeller Foundation and the Department of Health of the Commonwealth of Australia.

raised by the bird's struggles had settled, the cloth was removed and the contents of the cage were thoroughly drenched with antiseptic solution. At the *post mortem* examination the state of nutrition of the bird, the presence of cloacal soiling—evidence of diarrhœa—the size of the spleen, and evidence of past infection of the air sacs were particularly noted. Mouse inoculations were made from spleen emulsions.

The following method was employed to isolate the virus from sputum. After routine microscopic examination the sputum was ground with quartz powder and one or two cubic centimetres of broth. The emulsion was lightly centrifuged and the supernatant fluid was inoculated intraperitoneally into mice. As a rule three mice were inoculated with 0·5 cubic centimetre of the emulsion and three with 0·05 cubic centimetre, the smaller dose being given to increase the chance that the mouse would resist bacteria of moderate virulence which might also be present in the sputum. The stage at which the mice were killed varied according to circumstances, but if no symptoms appeared it was usually between ten and fourteen days. As a rule, positive results could be recognized in the mice receiving the primary inoculum by the presence of typical L.C.L. bodies in smears from the peritoneal exudate or spleen. In the case of the sputum of the first patient reported, one of the mice showed a greatly enlarged spleen and slight peritoneal exudate, but no L.C.L. bodies were found. Subinoculation of spleen emulsion to mice gave typical illness, and in these mice the characteristic signs were present and L.C.L. bodies could readily be demonstrated. Smears were stained by Castaneda's method.

Human Infections.

During the past six months seventeen cases of probable human infection by psittacosis have been recognized in the State of Victoria. These may be divided into two groups according to the source of infection, whether (*a*) aviary-bred birds or (*b*) birds recently captured in the wild. In the first group of two patients the infecting agents were budgerigars (*Melopsittacus undulatus*).

Group (a) Infections.

CASE I.—In November, 1935, Dr. G. H. Kennedy, a physician practising in a country town, reported the first suspicious case, that of a woman of thirty-five years, who had kept for several years an aviary of 70 to 100 birds—finches, cockatiels and budgerigars. About one month prior to the onset of her illness she had purchased six budgerigars from a bird dealer at a market in Melbourne. These birds had died within four weeks of their purchase. Two cockatiels (*Leptolophus hollandicus*), aviary-bred birds, three years old, and previously healthy, which had been placed next to the new birds in her aviary, sickened and died shortly before the onset of her illness on November 16. The details of the patient's illness have been published in full by Dr. Kennedy.[2] The clinical picture conformed to the description of the disease as seen in other countries. A specimen of her sputum was obtained on the fourteenth day of her illness and sent to the institute. The presence of the virus of psittacosis therein was established by the method previously detailed. On the fifteenth day of her illness four budgerigars from the patient's aviary were sent alive to the institute and there killed. In two birds slight thickening of the air sac membranes was observed and enlargement of the spleens. The spleens were pooled and mice inoculated with emulsions prepared therefrom. Both mice became sick in nine days and L.C.L. bodies were seen in smears prepared from their spleens. The liver of one mouse was inoculated into two more mice. Both these mice developed psittacosis in four days. Thus the physician's diagnosis was confirmed by the detection of the virus of psittacosis in the sputum of the patient and in two of four apparently unaffected budgerigars of her aviary.

CASE II.—Early in March, Mr. M., aged fifty years, living in Melbourne, became ill. His sickness was suspected first of being influenza. Later, typhoid or undulant fever was suggested. When extensive signs of lung involvement were discovered on the tenth day of his illness, the statement of his relatives that he had been closely associated with parrots was considered and sputum was sent to the institute for examination. The virus of psittacosis was proven to be present therein. All the budgerigars of his aviary, fourteen in number, some of them recently purchased from bird dealers, and one rosella (*Platycercus eximius*) were destroyed and examined. Several birds, including the rosella, showed enlarged spleens, but no virus was isolated.

The source of infection in this case must be considered uncertain.

In these two case, like most of those reported recently from California[3] and from Germany,[4] infection has apparently taken place from aviary-bred budgerigars amongst which the disease was present in a relatively mild form. We have had no opportunity to investigate the conditions in Australian budgerigar breeding establishments, but these two occurrences suggest strongly that the extent of mild psittacosis infection in them is not very dissimilar to that found in California.

In the remainder of the cases which we have to discuss infection was contracted from recently caught wild birds; and they exemplify the most important aspect of the epidemiology of human psittacosis in Australia. One of this group of fifteen patients was infected by a galah (*Kakatoe roseicapilla*) and two by Murrumbidgee smoker parrots (*Barnardius* sp.)

Group (b) Infections.

CASE I.—Early in January, Mr. O'M., aged fifty years, had purchased a galah (*Kakatoe roseicapilla*) from a dealer in the market in Melbourne. The bird was one of a number of young galahs caught in the country and sent in a crate to Melbourne. The galah died at the end of January. Early in February, Mr. O'M. developed a severe infection—typical psittacosis. The presence of virus in his sputum was detected by the method already described.

CASE II.—In a country town near the Murray River, on February 1, Mr. G., aged forty-nine years, purchased two Murrumbidgee smoker parrots (*Barnardius* sp.) which had been caught in nets across the river in New South Wales. One of these birds died on February 28, ten days after the onset of Mr. G.'s illness. Mr. G.'s illness was severe and typical of psittacosis, and the presence of virus in the sputum of the patient was proven. The second parrot was destroyed and forwarded to the institute for examination. It showed signs of active psittacosis, and mice inoculated with an emulsion of the spleen died with typical symptoms and *post mortem* signs.

CASE III.—On March 10 the daughter of Mr. G. became ill with fever, severe backache, headache and drowsiness. Her fever subsided after ten days. One scanty specimen of sputum was obtained from this patient, but the virus of psittacosis was not detected.

A group of twelve cases was reported in December, 1935. All the patients had been infected from one batch of recently caught white sulphur-crested cockatoos (*Kakatoe galerita*). An account of the circumstances of this outbreak will therefore be given in some detail.

In the survey of wild parrots reported last August, twelve individual white cockatoos from the Kerang district had been examined. In eight of these birds the spleens were normal in size; in four the spleens were enlarged, but the presence of virus could not be demonstrated.

A district less than one hundred miles from Melbourne, near Kyneton, has been for many years the home of flocks of these cockatoos. The country is open grazing country, lightly timbered with tall specimens of eucalyptus. In the spring the cockatoos nest in hollows in these trees. Rarely are more than three young birds found in one nest, the usual number being one or two. During October and November several men in the district climb the trees, by means of ropes, to the nests, which may be as high as seventy feet. If they judge the birds to be old enough to leave the parents, the bird-catchers lift them from the nest by a loop of wire, bring them to the ground and send them in crates to dealers in Melbourne. The birds are sought after by dealers to meet the demand for them as pets. When they have become old enough to feed themselves, they sell readily, particularly to the residents of the poorer suburbs. It is not usual to keep a pet cockatoo in a cage. Until he is accustomed to the home, his movements are restricted by a long chain which attaches him to his perch. Later he is allowed the freedom of the allotment or is carried about on his owner's shoulder. This freedom and the ability to learn a limited vocabulary have made the cockatoo popular. In the crates which convey them to Melbourne they are crowded together, and on arrival several in each crate may be dead. In the 1935 season a few young birds were found dead in the nests. This occurrence was attributed by the bird-catchers to desertion by the parents after inspection of the nest by another man. One of these men, F.B., was interviewed. He considered that his business was the smallest in the district, and he named four other local men engaged in the trade; yet during the 1935 season he had taken from the nests 289 young cockatoos. He sold four locally for four shillings each. These remained well. He sent 133 to one bird dealer in Melbourne, 30 to another, and 57 to a third. Two of these dealers are well established, and their birds are kept clean and adequately fed. We were unable to trace any instance of human infection from birds retailed by these dealers, though no systematic search could be made. Unfortunately, in Victoria there is no system comparable with that in California, of compulsory recording of the names of purchasers of parrots.

F.B. sent a fourth group of 49 cockatoos to Mr. X., who lived in an industrial suburb of Melbourne. Mr. X. is not an established bird dealer. He does other casual labouring work, but each season has obtained some cockatoos from the country. These birds were fed by his wife and son, aged twelve, and were kept penned in a small, dark shed in his back yard. The group of 49 birds had been sent by F.B. to Mr. X. in four consignments between November 8 and December 3. On November 28, Mr. X. wrote to F.B. for advice for treatment of sick birds. On December 16, Mr. X. stated that of the 49 birds, eight or nine had died, seven had been sold to different people in the neighbourhood, and about twenty had been taken by his son, aged twelve, to be sold on the kerb at the market. Eleven cockatoos, obviously sick birds, were destroyed on December 16 and four were examined. The *post mortem* appearances were practically uniform in all four birds. There was considerable emaciation with atrophy of the pectoral muscles and cloacal soiling. The livers were enlarged and greenish-brown in colour. There were active inflammatory changes with purulent lymph on the walls of the anterior air sacs. The pericardium was similarly coated and contained turbid fluid. Direct smears of pericardial exudate from two cockatoos contained enormous numbers of L.C.L. bodies, and spleen emulsions from all were rapidly fatal for mice, with typical findings. The cockatoos were thus actively infected with psittacosis and must undoubtedly have been excreting large amounts of virus.

Figure I shows the instances of human infection traced to cockatoos kept and retailed by Mr. X. On December 1, his son, aged twelve years, developed a mild infection. On December 3, his wife, aged thirty years, became severely ill with typical psittacosis. On December 16, his daughter, aged three years, developed a mild infection and quickly recovered. On December 13, his mother-in-law, aged fifty-eight years, became severely ill. She had been in close contact with her daughter in hospital, but she had not visited the house where the cockatoos were kept after December 3.

During the last week of November, Mr. X. sold a cockatoo to a woman, aged fifty-six years, Mrs. C., who lived in the neighbourhood. This bird died on December 11. Three cases developed in three members of this household: Mrs. C. on December 1, Mr. C., aged fifty-six years, on December 11 (both moderately severe infections), while the son of the house, aged eighteen years, developed a milder infection on December 23.

On December 1, Mr. X. sold a cockatoo to Mrs. McD., a neighbour, aged sixty-two years. The bird died eight days later. After the introduction of this bird to Mrs. McD.'s house, four of her parrots died. Mrs. McD. developed a severe illness on December 6. On the twelfth day of her illness a specimen of sputum was obtained for examination. The presence of the virus of psittacosis therein was established by methods already described. On December 20 her daughter-in-law, who lived in the house, developed a mild infection. On November 19, Mr. X. sent his son, aged twelve years, to the market with a number of cockatoos, in order that the boy might sell them to passers-by. On that date, Mr. Co.,

aged fifty-six years, who lived in a distant suburb, purchased two young cockatoos from a lad about twelve years old, who was selling birds on the kerb of the market. We have not been able to establish definitely that this lad was the son of Mr. X., but we are reasonably certain. On November 28, Mr. Co. developed a severe illness typical of psittacosis. On December 15, his daughters, aged twenty-four and twenty years respectively, developed sickness of moderate severity, but of shorter duration than their father's illness. The two cockatoos purchased on November 19 were the only pets kept at the home of Mr. Co. These birds were destroyed on December 24 and examined. It was proven that one had active and the other subacute psittacosis.

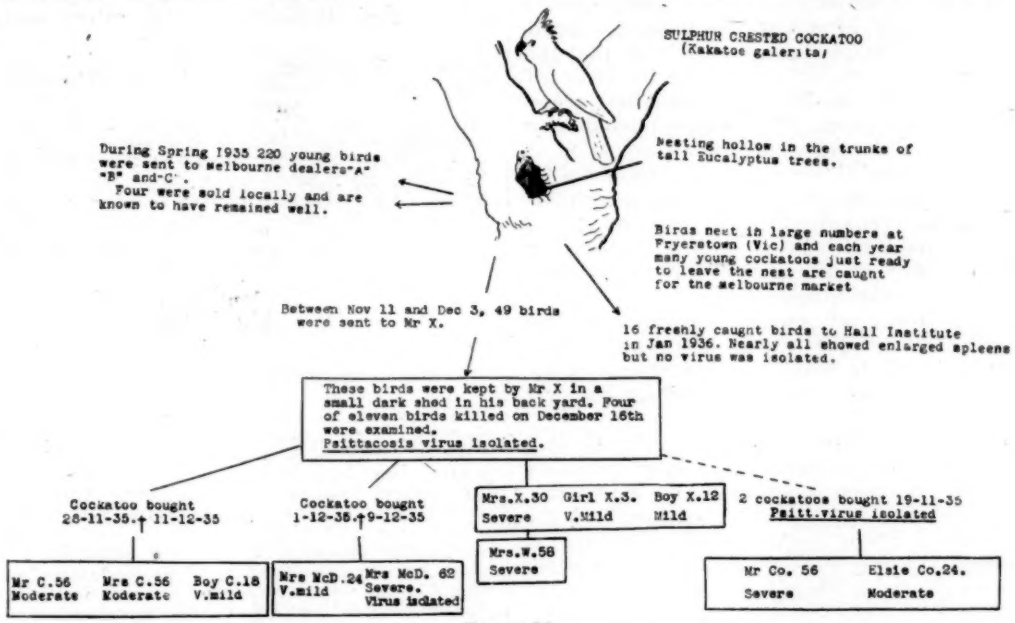
SULPHUR CRESTED COCKATOO
(Kakatoe galerita)
During Spring 1935 220 young birds were sent to Melbourne dealers "A" "B" and C.
Four were sold locally and are known to have remained well.
Nesting hollow in the trunks of tall Eucalyptus trees.
Birds nest in large numbers at Fryeretown (Vic) and each year many young cockatoos just ready to leave the nest are caught for the Melbourne market
Between Nov 11 and Dec 3, 49 birds were sent to Mr X.
16 freshly caught birds to Hall Institute in Jan 1936. Nearly all showed enlarged spleens but no virus was isolated.
These birds were kept by Mr X in a small dark shed in his back yard. Four of eleven birds killed on December 16th were examined.
Psittacosis virus isolated.
Cockatoo bought 28-11-35.† 11-12-35
Mr C.56 Moderate
Mrs C.56 Moderate
Boy C.18 V.mild
Cockatoo bought 1-12-35.†9-12-35
Mrs McD.24 V.mild
Mrs McD. 62 Severe. Virus isolated
Mrs.X.30 Severe
Girl X.3. V.Mild
Boy X.12 Mild
Mrs.W.58 Severe
2 cockatoos bought 19-11-35 Psitt.virus isolated
Mr Co. 56 Severe
Elsie Co.24. Moderate

FIGURE I.[1]

To illustrate the history of a small epidemic in Melbourne due to sulphur-crested cockatoos. The sign † indicates death of the cockatoo.

Clinical Findings.

Detailed clinical descriptions of seven of the twelve cases associated with cockatoos have been reported by the attendant physicians, Dr. F. C. Ross and Dr. L. Rabinov.[5] Clinically, all the seventeen infections resembled those described in other countries, with the exception that diarrhœa was a frequent symptom in the early stages of the illness of those patients who had been associated with cockatoos. Fever, headache, cough, drowsiness and diarrhœa were the most common symptoms. A feeling of well-being, despite severe illness, a pulse rate slower than that anticipated from the temperature and degree of toxæmia, signs of extensive lung involvement but scanty sputum were characteristic signs. In the early stages these patients caused their physicians to suspect influenza or typhoid fever, but the development of signs of bronchopneumonia led to inquiries for another diagnosis. In patients over thirty years the illness was more severe and protracted than in the younger patients. All patients recovered.

Discussion.

The chief interest of these cases from the epidemiological point of view centres round the development of active and infectious psittacosis in recently caught young cockatoos. It will be remembered that in our survey of wild parrots in the summer of 1934-1935 a certain number of cockatoos were examined. Of three batches of galahs (*Kakatoe roseicapilla*) examined, two from northern Victoria contained twenty-five birds with normal spleens and no evidence of psittacosis, and two birds with enlarged non-infective spleens. A batch of six from southern Queensland, on the other hand, all had enlarged spleens, one of which contained active virus. Among twelve sulphur-crested cockatoos from Victoria were two birds with typical enlarged spleens but no demonstrable virus. The others were normal.

When the association of sick cockatoos with the December outbreak of human infections had been demonstrated, it became important to determine the incidence of psittacosis in the wild cockatoos in the region from which the infected birds had been captured. From the same bird-catcher (F.B.) sixteen young cockatoos were obtained. Ten of these had been taken from the nests the preceding day and six about five days before examination. They were all well nourished and apparently full-grown. Nearly all had distinctly enlarged spleens and

[1] One case was inadvertently omitted from the diagram—that of Hilda Co. 20 (moderate). It should appear beside "Elsie Co. 24".

several showed moderate thickening of the air sac membranes. On the macroscopic appearances we were confident that the virus would be isolated from most of the spleens, but in fact none were infective for mice.

Combining all these findings, we are led to believe that infection with psittacosis is very frequently contracted by young cockatoos in the nest, but is usually overcome by the time they are captured, at least in so far that the spleen no longer contains virus. Unfortunately, we did not examine the kidneys of the cockatoos whose enlarged spleens were non-infective, for, as Meyer has suggested, it is possible that the virus persists longer in the kidney than in the spleen. The frequency with which we have observed cockatoos of both species which some weeks after capture have developed active psittacosis, makes it highly probable that the infection is of intrinsic origin. Under the crowded conditions existing in Mr. X.'s premises cross-infection from one to the other would undoubtedly occur and might be responsible for a proportion of the infections; but it seems more reasonable to regard the unhygienic conditions under which his birds were kept—crowding, lack of sunlight and of cleanliness, malnutrition—as acting rather by diminishing the resistance of the birds to the virus lying dormant in their tissues.

From the point of view of controlling the incidence of human psittacosis from this source, it seems advisable to make the following assumptions, although their complete proof is not yet available.

Practically all cockatoos are infected in the nest with psittacosis virus which normally produces little in the way of symptoms and no visible anatomical change beyond an enlargement of the spleen. If the young cockatoo, after capture, is kept under good conditions, it remains healthy and presents no danger to human beings. When, on the other hand, birds are crowded into small spaces, with inadequate food and sunlight, their latent infection is lit up. The virus multiplies in many organs and is excreted in large amounts. Should such a bird come into close and repeated contact with a person over forty years of age, a severe attack of psittacosis is likely to occur.

Conditions in regard to other species of wild parrots seem to be broadly similar, but in Australia the cockatoos are the only species which have been clearly incriminated as an important cause of human psittacosis. Administrative action either to prohibit the sale of young cockatoos or at least to insist that they are kept under decent conditions seems definitely called for, but it is doubtful whether public interest is yet sufficiently active to bring this about.

Summary.

1. During the summer of 1935-1936, seventeen instances of probable human infections with psittacosis have been recognized in Victoria.

2. Psittacosis virus has been demonstrated in the sputum of five of these patients.

3. The most important sources of infection in Victoria are young newly captured birds, particularly cockatoos.

4. The evidence suggests that maintenance of birds under crowded, unhygienic conditions results in a lighting up of their naturally acquired infection with the virus.

5. Two proven human infections were apparently derived from aviary-bred budgerigars.

Acknowledgements.

We are indebted to Dr. G. H. Kennedy, Dr. F. C. Ross, Dr. L. Rabinov, Dr. J. P. Major, Dr. L. Hurley, Dr. V. T. O'Grady and Dr. W. Pook for allowing us access to their cases and for permission to refer to them in this communication, and to Miss Dora Lush for carrying out much of the laboratory work.

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Reviews.

SOME PROBLEMS OF GENERAL PRACTICE.

"THE MINOR MEDICINE OF GENERAL PRACTICE", by L. V. Snowman, is the latest volume of "Pocket Monographs on Practical Medicine".[1] It is a good book. In 101 pages the author discusses various problems of general practice, the captions ranging from "Rheumatic Affections", "The Alimentary System" to "Minor Cardiac Disorders", "Headache", "Diet" and others. His style is brief and refreshing, his English without fault, his views balanced. His judgement has been built on a broad foundation, with due regard to medical, surgical and psychological view points. For example, he warns us of the almost certain secondary malignant deposit in the spine in the case of sciatica in a patient from whom a primary malignant growth has been removed.

Again he writes: "A constipated patient who is ill enough to come to the doctor with his complaint is ill enough to warrant proper investigation"; and he urges the physician to try to exclude definitely bowel carcinoma as a possible cause of the constipation.

Chapter IV, "The Neurotic Subject", merits reading by any practitioner, general or specialist. Concerning anxiety states, Snowman refers to unsatisfied sexual desires as possible ætiological factors, but does not lean solely on the mephitic ideas of those soul-strippers who today see signs of phallic worship in any neurasthenic patient who may like to handle yacht tillers or golf clubs. "History taking is practically a therapeutic weapon with these patients and must be so exhaustive that any present or past source of worry will be elucidated." His views appeal to us, for we have noted in private conversation that most open-air-living Australian doctors (and patients) do not lay so much stress on sex matters as would seem to obtain in other countries. There is a proverb in Spanish: "*Habla poco y bien y tenerte han por alguien*" (speak little and well and you will be considered as somebody). This is applicable to Snowman in this minor work.

[1] "The Minor Medicine of General Practice", by L. V. Snowman, M.A., M.B., M.R.C.P.; 1936. London: John Bale, Sons and Danielsson. Foolscap 8vo, pp. 110. Price: 2s. 6d. net.

The Medical Journal of Australia

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AN INTERMEDIATE MEDICAL EYE SERVICE FOR VICTORIA.

DURING recent years one of the most pressing aspects of the hospital problem in Melbourne has been the overcrowding of the ophthalmic out-patient departments in the several public hospitals. The patients needing attention have become so numerous that in some places great difficulty has been experienced in providing it. These patients (and indeed those of any specialist outdoor department) are of three grades. The first comprises patients who cannot afford to pay any fee; they are those for whom public hospitals were originally intended and for the treatment of whom medical practitioners gladly give their services. A second group, small perhaps, but growing in number, consists of those who can well afford to pay the fees of specialists but who cry poor. The third is the large intermediate group of persons who cannot pay the full fees of private specialist practice, but who can pay something for what they receive. There are many in the last-mentioned group who, not wishing to be treated for nothing, will gladly pay a modified fee. In these circumstances it is not surprising that for some time past the members of the Ophthalmological Section of the Victorian Branch of the British Medical Association have felt that steps should be taken to establish an intermediate medical ophthalmic service in Melbourne.

On July 1, 1936, the clinic of the Medical Eye Service of Victoria was officially opened by the Honourable Sir Stanley Argyle. Sir Stanley Argyle was introduced by Dr. C. M. Gardner, Vice-President of the Ophthalmic Association; and Dr. Walter Summons, President of the Victorian Branch, and Dr. J. Newman Morris, Chairman of Council of the Branch, also addressed the gathering. The Victorian Eye Service is to all intents and purposes a copy of the organization that has been established in New South Wales for about two years. It is administered by a new body, the Ophthalmic Association; and under the Articles of Association any member of the British Medical Association who is a full-time practising ophthalmologist may become a member. The Ophthalmic Association is controlled by a council of five; four councillors are elected by the members of the Ophthalmic Association and one is nominated by the Council of the Victorian Branch of the British Medical Association. The Victorian Branch has thus given its full approval to the movement. The persons eligible for treatment are those whose total family income does not exceed five pounds per week. A comprehensive fee of fifteen shillings is charged, and for this sum the patient is examined and receives a prescription for glasses, if required; if necessary, a mydriatic is used. The patient also receives such advice, prescription for drugs and simple operative treatment as can be given at a single consultation. When necessary, a report is sent to the patient's general medical attendant. If further consultation room treatment is required, it is continued at a rate of ten shillings a week. Spectacles are supplied when necessary, and in these circumstances an inclusive fee (for examination, advice, treatment and glasses) of from twenty-five to thirty-five shillings is charged, the variation being dependent on the type of spectacle frame selected.

Medical practitioners do not need to be reminded that in many cases defective eyesight is not due to

a refractive error and that the welfare of the patient will best be served if he is examined and treated by a skilled ophthalmic surgeon. It is to be hoped that the success achieved in New South Wales will be more than equalled by the Medical Eye Service of Victoria. Victorian medical practitioners will, if they are wise, use the new service on all suitable occasions, first of all because they will be doing a service to their intermediate patients, and secondly because they will be assisting in an attempt to solve at least part of the hospital problem.

Current Comment.

VITAMIN C.

THE clinical use of concentrated vitamins is apparently increasing rapidly, if we may judge by the profusion of literature and samples which are sent to medical practitioners regularly by manufacturing chemists. This form of therapy rests upon sound foundation in some cases; no better example could be found than rickets. But in many other conditions the reasons for its use, if any, have been vague, and it must be admitted that not infrequently medical practitioners prescribe high vitamin diets on general principles rather than because they have positive proof of the existence of a true deficiency state. Since the use of pure crystalline vitamin *C* (cevitamic acid) has been urged in certain conditions like purpura, on the grounds that it may not always be absorbed when taken by mouth in the usual way, it is of interest to inquire what proof is available of its absorption. Clinical proof is adequate if it is certain that the patient's condition is due to an avitaminosis, but measurement is preferable. Measurement can be accomplished, and a useful review of the methods employed by various workers during the past few years is given by J. B. Youmans, M. B. Corlette, J. H. Akeroyd and Helen Frank.[1]

These authors point out that normal persons of good nutrition excrete relatively constant quantities of cevitamic acid in the urine, that this excretion continues for some time if all supplies of vitamin *C* are cut off, and rises appreciably as soon as the intake is increased, thus indicating that the presence of a reserve supply in the body and a definite saturation point can be demonstrated. Working on these lines, they investigated 15 subjects whose diets were suspected of being inadequate in vitamin *C*, and 16 normal control subjects. The point with which we are concerned at the moment is that these workers consider that a daily excretion of 20 milligrammes of vitamin *C* in the urine may be regarded as normal, and an excretion of 30% of a test dose of about 600 milligrammes appears to be the lower limit of normal saturation. This figure agrees with the normal excretion rate arrived at by other investigators, such as M. A. Abbasy, L. J. Harris, S. N. Ray and J. R. Marrack.[1] In the opinion of H. E. Archer and G. Graham, expressed in a recent article, the most significant diagnostic test in scurvy is the observation of the amount of ascorbic (cevitamic) acid required to raise the patient's excretion rate above 75%.[2] Perhaps we may see before long the addition of these tests to clinical medicine, for we are beginning to suspect that a relative deficiency of the accessory food substances may not be uncommon and that it may give rise to numbers of vaguely understood forms of minor illness. An example of the possible importance of vitamin *C* in actual disease other than scurvy may be found in rheumatism. Though it must be regarded as unproven, there is a plausible hypothesis that rheumatic fever depends upon the combination of infection and vitamin *C* deficiency.

At the second annual meeting of the American Association for the Study and Control of Rheumatic Diseases a number of interesting papers were read. One of these, by James F. Rinehart, dealt with the study of vitamin *C* deficiency in rheumatic fever.[3] Rinehart carried out experimental work on guinea-pigs, which were maintained on a basic diet adequate in all respects except that it was devoid of vitamin *C*, and which were submitted to inoculation with strains of hæmolytic streptococci known to be pathogenic to this species. A difficulty is found in the fact that scurvy itself tends to produce arthropathies in these animals, but Rinehart's contention is that the deficiency state is a contributory factor in the ætiology of rheumatic fever. He has produced lesions both in the joints and in the heart, and in the latter, lesions are found both in the valves and the myocardium, suggestive of the typical changes seen in the human subject. Subcutaneous nodules were also produced, and the author comments on the fact that these are observed in both rheumatic fever and rheumatoid arthritis, diseases the similarities and differences of which form a fascinating study. He makes no claim that these experimental lesions are identical with those of human rheumatic fever, but thinks they are fundamentally similar. He also considers that multiple infective factors may be operative in acute rheumatism, and that these may produce their effect by toxic action or strictly local operation or by an allergic process. Allied to this, however, he believes, is a deficiency factor, and points out that epidemiological evidence points to the importance of a nutritional factor, and that individual capacity to store or utilize the vitamin is now established.

A number of papers have been written on another aspect of vitamin *C* deficiency, capillary fragility,

[1] *The American Journal of the Medical Sciences*, March, 1936.

[1] *The Lancet*, December 21, 1935.
[2] *The Lancet*, March 28, 1936.
[3] *The Journal of Laboratory and Clinical Medicine*, March, 1936.

which may apparently be regarded in some cases as a type of latent scurvy. J. S. Wright and A. Lillenfeld have summarized the position concerning cevitamic acid in this condition in a recent article.[1] They point out that the pure crystalline substance can be synthesized, and that it may be administered intravenously, although the problem of sterilization is difficult in a substance which is so readily affected by heat. Its value in scurvy is undoubted, but is doubtful in thrombocytopenic purpura or familial hæmophilia; it appears to be of service in the forms of purpura due to a capillary fragility. Wright and Lillenfeld assert that many cases of early and "subclinical" scurvy are missed, with which we would all agree; perhaps when we have some adjuvant test, such as referred to above, such conditions will be more easily recognized. They point out that the patients suffering from vitamin *C* deficiency are of two classes: those whose intake is inadequate and those who are unable to utilize the vitamin even from an adequate diet. It is for the latter class that parenteral administration may be needed. Dosage is not yet accurately settled: 1,000 milligrammes daily have been given intravenously over a period of fourteen days, a quantity far above the ordinary dose, which is usually placed at 100 to 150 or 200 milligrammes every day or two. For prophylaxis a daily dose of 20 to 30 milligrammes of cevitamic acid is adequate. Lest it should be thought that the diagnosis of minor degrees of capillary fragility is beyond the scope of the average medical observer, it should be noted that the most simple test is that of the tourniquet. To perform this on a clear area on the forearm a circle 2·5 centimetres in diameter is drawn. On this the petechiæ are counted, produced by applying a sphygmomanometer cuff on the upper arm for 15 minutes. A count of 10 is within normal limits; a count of over 20 is definitely abnormal, according to Wright and Lillenfeld; no standard is yet agreed upon generally, but pending more accurate methods this might be used as a rough standard.

RESUSCITATION OF THE NEW-BORN.

AT a recent meeting of the Royal Society of Medicine, N. B. Capon, of Liverpool, drew the attention of the meeting to the importance of neonatal mortality. In England and Wales the neonatal death rate in 1933 was about 50% of the total infant mortality rate. Capon pointed out that in the period from 1906 to the present day, while the infant mortality rate had decreased by about 50%, the neonatal death rate had decreased by only about 20%. John F. McGrath and Katherine Kuder[2] believe that many new-born babies are lost because of lack of proper treatment of *asphyxia neonatorum*. It is, of course, very difficult to estimate the frequency of asphyxial fœtal mortality. Cruikshank, in a Medical Research Council report published in 1930, recorded a very exhaustive investigation of *post mortem* examinations of 800 neonatal deaths. He found that 68% of these were due to *asphyxia neonatorum* and "allied conditions". Defining the neonatal period as fourteen days, McGrath and Kuder find an incidence of pathological respiratory conditions in 17·59% of 216 neonatal deaths. Yandell Henderson considers that stillbirths and neonatal deaths due to respiratory conditions amount to 4 in 100. The wide variation in these estimates seems to show that further investigation of *asphyxia neonatorum* is necessary.

McGrath and Kuder consider that the usual treatment of *asphyxia neonatorum* lags far behind modern knowledge of the physiology of respiration. There has been little effort to improve the method of accomplishment of normal breathing. Even modern text-books on obstetrics continue to devote space and pictures to demonstrate methods of treatment that are no longer suitable. Our knowledge of the physiology of respiration is still far from perfect, and there are still much doubt and controversy concerning the chemical conditions in the blood in the normal and the asphyxiated baby; but sufficient work has been done to improve on the resuscitation methods of twenty years ago. Henderson contends that the beneficial results of carbon dioxide administration demand its more universal use in aspyhxia, and, in spite of controversy, his advice cannot be ignored. He also points out that oxygen determines the sensitivity of the neuro-respiratory system to its specific stimulus, which is carbon dioxide. Extreme deficiency of oxygen in the fœtal circulation paralyses the neuro-respiratory system.

Moncrieff has emphasized the fact that many of the deaths during the first month of life may well justify the suggestion that the word "suffocation" is a fairer one than asphyxia. The terms asphyxia and atelectasis camouflage rather than define the true significance of the fœtal failure to breathe. There is no doubt that respiratory failure at birth is essentially the same, both in mechanism of production and treatment, as respiratory failure at a later period in life. When a new-born baby does not breathe normally, there is either a central anomaly, such as (*a*) immaturity, (*b*) intracranial injury or pressure, or (*c*) chemical depression due to narcotics, oxygen lack or carbon dioxide excess secondary to improper arterialization of the centre; or else there is a peripheral abnormality, such as immaturity of the lung alveoli, developmental deficiency, circulatory failure or the more common condition of obstruction somewhere in the air passages. It is with the last-mentioned group that these authors are most concerned; for therein, they believe, lies the greatest opportunity to lessen the incidence of neonatal death.

The frequency of obstructive material, mucus, amniotic fluid, meconium, *vernix caseosa*, or epithelial *débris* is evident to every obstetrician and is almost always found

[1] *Archives of Internal Medicine*, February, 1936.
[2] *The Journal of the American Medical Association*, March 14, 1936.

in careful microscopic study of infant lungs at autopsy, as in the report of Farber and Sweet, in which 88 per cent. of 124 unselected babies dying within the first five weeks of life showed aspirated material in the bronchioles and alveoli.

Admittedly *asphyxia neonatorum* is often unexpected and unpreventible. McGrath and Kuder consider, however, that proper *ante partum* and *intra partum* treatment can eliminate many of the causative factors.

The actual treatment of asphyxia is based on three fundamental concepts: First, open airways. That is, all obstruction must be removed; mucus, amniotic, meconium or other fluid must be removed by aspiration. Second, insufflation or distension of lung alveoli, which must be brought about. Third, stimulation of the neuro-respiratory centre with carbon dioxide after its sensitivity has been increased with oxygen.

The authors conclude that more careful study and recording of the factors relative to the incidence, causation and treatment of *asphyxia neonatorum* should be the continuous duty of every maternity service. In their opinion, the method of direct exposure, intubation, and insufflation of oxygen, 90% to 95%, and carbon dioxide, 5% to 10%, is the treatment *par excellence*, because of its ease of application and its beneficent results.

BOTULISM IN SHEEP.

AN investigation of considerable interest on botulism of sheep has been carried out in the Department of Agriculture, Western Australia, by E. J. Underwood and F. L. Shier.[1] The investigation was carried out during the summers of 1933-1934 and 1934-1935. In the first of these summers there was an abnormally early appearance of green feed; the second summer was long and severe. The objects of the investigation were threefold: in the first place to investigate the effects of supplementary feedings on the incidence and control of the pica which impels sheep to consume the carcasses of rabbits (the principal source of the toxin of botulism); in the second place to investigate the results of supplementary feedings on the growth and live weight of sheep; and thirdly to determine the effects of such feeding on the inorganic phosphorus levels of sheep.

During the dry summer months the live weight of the sheep fell considerably, and heavy supplementary feeding was necessary to prevent this loss of weight. The grazing at the end of the summer became dry and scanty and also low in protein and phosphorus. It was found that phosphorus deficiency was not the limiting factor in nutrition, for no increase in weight was brought about by a large intake of soluble phosphorus lick. Moreover, the levels of inorganic phosphorus in the blood of the sheep showed no significant fall, even during the late summer period, but remained at a constant level throughout the year. These findings are hard to understand, and Underwood and Shier suggest that the phosphorus requirements of sheep may be much lower than was previously supposed, or that other nutritive deficiencies have the effect of reducing the amount of phosphorus required in the diet. These hypotheses have been put forward by Marston, who, according to Underwood and Shier, also pointed out that the dry herbage may not truly represent the material actually consumed by sheep, since sheep display much selection in their grazing.

The pica of sheep in dry seasons is most interesting. They displayed a special liking for the fleshy portions of rabbit carcasses. They ate skin and viscera on occasions, but showed no interest in bones. A special taste for rabbit fæces was noted. The phosphatic lick that was consumed had no effect in preventing the pica, though a partial control was given by some of the food supplements. The pica disappeared with such remarkable rapidity on the advent of the first green feed, even when it was available in only very small amounts, that it seemed unlikely that it could be caused by a direct deficiency of an essential nutrient or nutrients. Underwood and Shier suggest that the pica may be due not to a direct deficiency of an essential nutrient, but to some food error such as would cause a disturbance of the acid-alkali balance, or to a physiological upset resulting from the dry and fibrous nature of the diet. The results of further investigations, promised by them, will be awaited with interest, for the subject has a bearing not only on the general causation of disease, but on one of the most important industries in Australia.

[1] *The Australian Journal of Experimental Biology and Medical Science*, June, 1936.

THERAPY AND THE NERVOUS LESIONS OF PERNICIOUS ANÆMIA.

THERE is no doubt that treatment of pernicious anæmia by parenteral injections of liver preparations is by far the best method of treating the nervous lesions of the disease. None can say whether this therapy has any specific action upon degenerative processes in the spinal cord, but, as Davidson has recently pointed out, the fact remains that in 70% of treated patients there is a symptomatic improvement in the nervous manifestations. For some undetermined reason the symptoms indicating lesions of the posterior columns are much more amenable to treatment than similar changes in the lateral columns. Thus patients who have suffered from long-standing ataxia may actually recommence to work efficiently while the signs of pyramidal degeneration still persist.

Edward S. Mills[1] has set down the results of liver therapy as affecting forty-five patients treated at the Montreal General Hospital during the past seven years. While the exact nature of the nervous lesions in pernicious anæmia is still, he writes, a

[1] *The American Journal of the Medical Sciences*, January, 1936.

matter of debate, it is generally conceded that the degenerative changes, if untreated, gradually reach the stage of sclerosis. Further, these changes are not found during the course of other hæmolytic anæmias; the cause of the pernicious anæmia and of the nervous lesions seems to be one and the same. Mills based his diagnosis of the well-known subacute combined degeneration of the cord upon deviations from normal of two-point discrimination and of the vibratory sense, for he found that all other sensory signs were fleeting, uncertain and indeterminate. He remarked, too, that in fewer than half of his cases was there any alteration in the tendon reflexes. A very interesting point in connexion with this aspect of the diagnosis is that while eight of Mills's patients suffered from an involvement of the lateral tracts of the cord, as shown by the presence of exaggerated tendon reflexes and of incoordination, yet the actual incidence of lateral tract affection may be much greater than these figures indicate. The reason is that degeneration in the lateral tracts results in the production of greatly increased muscle tone with a corresponding effect upon the tendon reflexes; the involving of the posterior columns, however, tends to neutralize or to nullify these effects. Coexistent disease in both lateral and posterior tracts may therefore give a clinical picture in which, while there is grave functional loss in movement and coordination as well as in sensation, the tendon jerks are normal.

Four patients in Mills's series are worthy of special note. Three of them suffered from psychoses, one was completely disorientated and had so intense a double papilloedema that the diagnosis of cerebral tumour was seriously discussed, and yet another with much the same cerebral signs had no papilloedema at all. The blood counts of these four patients showed that little or no anæmia was present, but in all there was macrocytosis. In all four, once the blood picture returned to normal, the signs of combined degeneration disappeared and the cerebral signs and symptoms vanished.

In order to assess the worth of liver therapy upon the nervous lesions of pernicious anæmia, Mills first considered those patients in whom the treatment had been inadequate, comparing their fate with that of others to whom a suitable preparation of liver had been given at proper intervals and for a proper length of time. The presence of macrocytosis was held to betoken the failure of treatment, as was any abnormality in the erythrocyte count; and apart from any indications derived from consideration of the blood picture, treatment was at once intensified if there were complaints of symptoms indicating damage to the nervous system. In the total number of forty-five, twenty-eight of the patients were held to have received suitable treatment. Seventeen unfortunates were classed as inadequately treated. Six of these latter are now dead, three are completely disabled, five are suffering from disabilities of greater or lesser extent, and only three can claim to be in all respects normal. The group of twenty-eight successfully treated patients is a happy contrast to all this. In half of these, roughly, the nervous lesions have improved and show no sign of progression, and a mere three are unfit to perform their daily work.

Little benefit may be expected to follow treatment if the patient is the subject of advanced heart disease or is senile; but in many others the effect of a weekly dose of five cubic centimetres of an active liver extract, injected into the muscles, is little short of miraculous. Lost knee jerks return after an absence of months or years, the Babinski sign may be replaced by a normal plantar reflex, and other evidences of damage to the pathways in the spinal cord may gradually become indistinct and disappear. The medical attendant of a patient with subacute combined degeneration of the cord should believe that the degeneration may exist without proceeding to the stage of actual destruction, and should hesitate to predict an unfavourable outcome in the case of any sufferer who shows the will to recover.

CHARLES BARRETT LOCKWOOD.

THOSE who graduated or were students of medicine at the dawn of this century are all familiar with the name of Lockwood. His students stood in awe of him, some of them feared the lash of his tongue, all respected him and many loved him. He was a man with a remarkable and forceful personality, who, with Butlin, fought the battle for antiseptic surgery when the purblind and the obstinate at "Bart's" refused to follow the teachings of Lister or followed them in a half-hearted way without understanding them. He was one of a type that is seldom seen nowadays. One of his students wrote of him: "No one was ever so rude as Lockwood. No one ever made a man feel so small, but he was a man, and we just loved him." These words are quoted in the introduction to a small brochure that has been awarded the Wix Prize for 1934 at Saint Bartholomew's Hospital.[1] As a teacher Lockwood was insistent on accuracy. Nothing was taken for granted; observation had to be keen and measurements exact. The eyes were used "first and much, hands next and little, tongue not at all". The best text-books, he used to say, were the patients in hospital. He used to have snippings taken from the skin of his own hands and from those of all his assistants in the operating theatre; these were incubated and a record was kept of the number of tubes in which microorganisms were grown. In this way he practised and taught asepsis and antisepsis. E. C. O. Jewesbury, who wrote this prize essay, has given a vivid account of Lockwood both as a teacher and as a man. Doubtless many present-day practitioners know nothing of Lockwood; we would advise them to read this booklet. They will find it both interesting and profitable.

[1] "The Life and Works of Charles Barrett Lockwood", by Eric C. O. Jewesbury, M.A., B.M., B.Ch. (Oxon); 1936. London: H. K. Lewis and Company Limited. Crown 8vo, pp. 112, with 12 illustrations. Price: Cloth, 3s. 6d. net; paper cover, 2s. 6d. net.

Abstracts from Current Medical Literature.

GYNÆCOLOGY.

Uterine Hæmorrhage.

W. M. WILSON (*Western Journal of Surgery, Obstetrics and Gynecology*, December, 1935) discusses some features of functional uterine hæmorrhage and considers that the incidence of hyperplasia of the endometrium in these cases is grossly over-estimated. The endometrium in the majority of the cases is composed of non-proliferating, non-secreting glands of the early or mid-interval type and a more or less œdematous hyperæmic stroma. This histological picture, together with the clinical evidence of hypoovarianism, suggests the term hypoplasia for this type of endometrium. It is also suggested that functional bleeding of this type may be due to a drop in the normal blood level of œstrin.

The Treatment of Leucorrhœa.

C. MENGE (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1935) discusses the ætiology and treatment of vaginal discharges. Excluding the cervix as an ætiological factor, he discusses the various theories regarding excessive vaginal discharge, and concludes that it is due in the majority of cases to a transudative diathesis. General treatment is important to diminish the excessive transudation, improve the quality of the blood, regulate the intestinal tract and stimulate the endocrine organs so that eventually a better physico-chemical structure of the serum in the connective tissues can be formed. To this end he gives calcium, iron and arsenic preparations and vitamins. Local therapy is indicated if there are any changes in the vaginal walls. He prefers a 2% to 3% watery solution of silver nitrate applied on tampons after all secretion has been removed. This can be combined with douches of zinc chloride or copper sulphate solution. If this fails, the trichomonas should be sought for as a secondary cause.

Results Following Unilateral Oophorectomy.

H. RUNGE (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1935) has investigated the results following removal of one ovary. His interest was aroused after observing a case of severe menorrhagia following a sterilizing operation, when one cystic ovary was removed with the tube on that side. In his opinion, the *corpus luteum* was removed with the cystic ovary. Analysis of a series of cases showed that regular menstruation was the rule in two-thirds. Amenorrhœa or scanty periods following operation points towards an ovary of poor quality, especially if obesity occurs showing marked endocrine deficiency. Excessive bleeding may be caused by too frequent ovulation or else there is a functional muscular weakness of the uterus. The typical change, however, after oophorectomy is cystic degeneration in the remaining ovary. If the ovary be matted in adhesions, it forms retention cysts. There need not be any menstrual irregularity in such cases. Occasionally a glandular cystic hyperplasia of the endometrium results from excessive ovarian stimulation. The author believes that whenever possible ovaries should be resected and not completely removed.

Treatment of Endocervicitis.

L. S. KRITSCHEWSKY AND E. WERBATUS (*Monatsschrift für Geburtshülfe und Gynäkologie*, March, 1936) discuss the ætiology and treatment of cervical erosion and endocervicitis. They describe the effect of colloidal solutions on the cervical mucosa and give the results following the use of a 1 in 20,000 ammoniacal silver preparation. Ten cubic centimetres were injected with a Luer syringe into the cervical tissues and repeated within five days over a course of two to eight injections. The injections were practically painless and all the patients remarked on the improvement in general health felt after the first injection. The average length of treatment was three weeks. In some cases pregnancy followed a period of sterility or former dysmenorrhœa and menorrhagia.

Treatment of Amenorrhœa.

J. ROCK AND M. K. BARTLETT (*American Journal of Obstetrics and Gynecology*, April, 1936) have investigated the result of treatment with "Antuitrin-S" of four married women who suffered from amenorrhœa for one to two and a half years. A dose of 500 rat units was given intramuscularly on alternate days till a total dose of 1,500 to 3,500 units had been given. Biopsies of the endometrium were made sometimes before and sometimes during treatment and in each case after treatment; in only two instances was there any change which would suggest increased ovarian function. They conclude that moderate doses have no ovary-stimulating effect, and they quote Johnson, who was unable to detect any effect on monkeys, though much larger doses were given.

Trichomonas Vaginalis.

J. L. COLLIS (*The Journal of Obstetrics and Gynæcology of the British Empire*, February, 1936), working at the venereal clinic of the General Hospital, Birmingham, has investigated the occurrence of vaginitis due to *Trichomonas vaginalis*. After reviewing the life history of the organism and describing the type of vaginitis which it causes, he states that the proportion of cases of persistent vaginal discharge in which the trichomonas is present varies from 40% to 60%. In his own clinic the percentage is 37·04. He considers that the trichomonas may travel as spores and lodge in the female vagina at various times, but will not cause any vaginitis until such time as the resistance of the vagina is lowered either by local or general disease. The condition is due to the *Trichomonas vaginalis*, but the free growth of this organism depends to some extent on the growth of other organisms. In the author's experience it is best treated by "Devegan" inserted in the form of tablets. Two tablets are inserted each day at the beginning of treatment. This is continued daily until the discharge is well controlled. Then one tablet a day is used for six weeks, after which one tablet a week is used until the organism has disappeared.

Mechanics of Uterine Support and Position.

WILLIAM F. MENGERT (*American Journal of Obstetrics and Gynecology*, May, 1936) has studied the mechanics of uterine support and position on eight cadavers. A weight of one kilogram was attached over a pulley by a tenaculum onto the lip of the *cervix uteri*. The structures attached to the uterus were then severed and observations were made on the subsequent descent of the uterus. It was found that a section of the round, ovarian, infundibulo-pelvic and the upper third of the broad ligaments hardly affected the position of the uterus in the pelvis. No incision was made in the pelvic floor, but this did not hinder experimental prolapse of the uterus. When section was made of the lower two-thirds of the broad ligament and the upper two-thirds of the paravaginal tissue, there was an average uterine descent of 10·5 centimetres. It was found that as long as any part of the upper two-thirds of the paravaginal and/or the lower two-thirds of the parametrial tissues were not cut, there was no marked descent of the uterus to a degree which would be considered to be actual prolapse. In the opinion of the author the paravaginal tissue seems to be more important than the parauterine tissue in the prevention of prolapse. These tissues have been described by various authors as ligaments or as sheaths of fascia, but it would not seem to matter very much as to the actual description. The point at issue is the amount of support that they give to the *cervix uteri* and to the upper part of the vagina.

Effect of Total Hysterectomy upon the Ovary of Macacus Rhesus.

THOMAS H. BURFORD AND ALBERT W. DIDDLE (*Surgery, Gynecology and Obstetrics*, April, 1936) have studied the effect of total hysterectomy upon the ovary of *Macacus rhesus*. Their object was to ascertain the relation between the ovary and the uterus in

endocrine function. After investigating the literature on studies on the human ovary after hysterectomy, they carried out a series of experiments on five female monkeys on which a total hysterectomy was performed, and the effect on the ovary was studied during a period of 121 to 217 days. Evidence was sought by vaginal lavage studies, by sex, skin colour records and examination of the ovaries at subsequent operations. A *post mortem* census of the follicles in the ovaries showed no increase in follicular atresia. The authors concluded that hysterectomy in this monkey was without effect on the ovary, except for purely traumatic effect. The monkey offers no evidence that the uterus has any endocrine influence upon the ovary. One monkey arrived at sexual maturity 103 days after removal of the uterus. Two other animals in the immature group did not reach sexual maturity at a later date, but there was no significant reduction in the size of the ovaries and no other gross change could be detected.

OBSTETRICS.

Gastric Analysis During Pregnancy.

H. WINKLER (*Monatsschrift für Geburtshülfe und Gynäkologie*, September, 1935) has investigated the chemistry of the gastric secretion during pregnancy, especially in cases of hyperemesis. Pregnant women without any previous gastric trouble show an increased total gastric acidity as well as free hydrochloric acid. The secretion curve is steeper than normal and the emptying time for the stomach is at the upper bounds of the normal. In more than half the cases a supersecretion exists. In the vago-hypertonic cases of vomiting, which usually are controlled by sedatives, these rates are greatly increased. With true toxic hyperemesis there is a marked lowering of these rates. He considers that this effect is produced by toxic effects on the autonomic nervous system, particularly through the parasympathetic nerves.

Vitamin E and the Anti-Proteolytic Factor in the Serum of Aborting Women.

EVAN SHUTE (*The Journal of Obstetrics and Gynæcology of the British Empire*, February, 1936) discusses the relation of deficiency of vitamin *E* to the anti-proteolytic factor found in the serum of aborting women. In previous papers the author had shown that serum of 70% or more of women aborting spontaneously displayed a characteristic resistance to proteolysis when exposed to tryptic solutions. The resistance, it was further shown, appeared to be due to an agent contained in the placenta and allied to œstrin. From the results obtained in the present paper, it is shown that a preparation of vitamin *E* (wheat germ oil) will temporarily remove the anti-proteolytic factor, and this property of vitamin *E* was constant in all the cases in which it was used. Further, there is a relation between the dose of vitamin *E* and the disappearance of the anti-proteolytic factor. Clinically, a suitable dose of vitamin *E* was found to be 48 cubic centimetres (12 drachms) of "Viteol" (wheat germ oil) on the first day, and 4 cubic centimetres (one drachm) on the subsequent days. This was sufficient to produce a cessation of symptoms of threatened abortion. The author concludes that vitamin *E* and œstrin, or a substance much like it, exist in equilibrium during pregnancy. If there is too much of the œstrin-like substance, the pregnancy is interrupted. An excess of vitamin *E* appears to neutralize this action, but has no effect itself on the pregnancy.

Wheat Germ Oil (Vitamin E) Therapy in Obstetrics.

E. M. WATSON AND W. P. TEW (*American Journal of Obstetrics and Gynecology*, February, 1936) report the treatment of one hundred patients with wheat germ oil for repeated abortion and threatened abortion. The patients were subdivided into four groups. The first group comprised those who had suffered two or more abortions; the second group had had one abortion; the third group had had threatened abortions; and the fourth group was sterile. The successful results were in the neighbourhood of 75%, but in sterility the therapy was found to be useless. The authors are of the opinion that whilst wheat germ oil is not a panacea for abortions in women, it is a useful weapon in the treatment of this condition, but is of no avail in the treatment of sterility.

Puerperal Infection due to Anaerobic Streptococci.

O. H. SCHWARZ AND T. K. BROWN (*American Journal of Obstetrics and Gynecology*, March, 1936) issue a further report on their investigation of the presence of anaerobic streptococci in puerperal infection. Since anaerobic streptococci are present in the vagina of about 40% of women, it can be appreciated that this factor is one of the main causes of endogenous infection, which, however, develops only when conditions such as trauma, shock and loss of blood favour their growth. Although infections which are due to ordinary pathological organisms are to a large extent within control, endogenous infection offers a more difficult problem. The authors, to overcome this difficulty, have used an instillation of 1% neutral acriflavine in glycerine and they have been able to halve their morbidity rate. Their report, which concludes ten years' study, confirms all the contentions of Schottmüller. They believe that when these facts become more generally appreciated and further knowledge is gained in the diagnosis and treatment of anaerobic infections, the name of Hugo Schottmüller will take equal rank with that of the great Semmelweis.

The Effect of Camphor in Oil on Lactation.

M. D. KLEIN (*American Journal of Obstetrics and Gynecology*, May, 1936) has found that intramuscular injection of camphor in oil is an effective method of treatment for the relief of breast engorgement. In ninety cases studied, camphor in oil, given intramuscularly, was found to inhibit lactation. The sooner, however, the injections were given *post partum*, the more effective was the action. The drug has an advantage over binders *et cetera* in an attempt to cause a regression to the non-lactating phase, which usually took only two to three days instead of the usual four or five. Two doses of camphor in oil, 0·09 gramme (one and a half grains) each, were given on the first day. One injection of 0·09 gramme was given daily for three successive days, making the total number of injections five.

Ætiology and Frequency of Premature Birth.

E. FELS AND H. SANDBERG (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1935) discuss the ætiology and frequency of premature birth at the Breslau Clinic. The percentage had decreased to 8·1, as compared with 11% to 13% in former years. This has been mainly due to more intensive antenatal supervision and the better treatment of syphilis. At the same time more pregnancies have been terminated for various conditions before fœtal death ensued. With this improved maternal supervision there is also a better knowledge of the causes of premature birth. The authors were able to classify two-thirds of their cases. As mass statistics are published the percentage of unknown causes of premature birth should steadily decrease.

Anæsthesia in Toxæmia.

H. WINKLER (*Monatsschrift für Geburtshülfe und Gynäkologie*, October, 1935) discusses the effect of chloroform anæsthesia given *à la reine* in the second stage of labour in cases of preeclamptic toxæmia. His review is mainly concerned with the ill-effects produced in a heart the myocardium of which has been grossly damaged by the toxæmia of pregnancy. The details of seven cases are given. Chloroform was administered at the end of labour and signs of cardiac failure were noted after delivery. The author considers that when the pregnancy is terminated in such cases of preeclamptic toxæmia, either by section or by induction of labour, the anæsthetic chosen should be gas and ether, neither of which has any effect on a heart which is already damaged.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held in the Anatomy Lecture Theatre, University of Adelaide, on March 26, 1936, DR. D. R. W. COWAN, the President, in the chair.

Some Disturbances of the Circulatory System.

DR. E. F. GARTRELL read a paper entitled: "Some Disturbances of the Circulatory System due to Pneumonia and other Toxic Conditions" (see page 76).

DR. K. S. HETZEL said that Dr. Gartrell had chosen wisely in selecting this subject for discussion. The conditions of which he had spoken concerned all medical practitioners, most of whom no doubt had formed definite opinions gained from personal practical experience.

Dr. Hetzel referred to the effect of toxæmia on the heart itself. Years previously many had held the opinion that the return to sinus arrhythmia in the heart of a child suffering from the toxic effects of diphtheria was of good prognostic significance and tended to show that the neuromuscular mechanism was coming back to normal. But were medical practitioners justified in assuming that at the present time? Certainly as far as rheumatism was concerned, they were sure that there could exist a smouldering carditis with sinus arrhythmia and a normal electrocardiographic tracing. In adults and the febrile patients with some toxic myocarditis sinus arrhythmia was not seen, for the heart rate was rapid.

In the peripheral vascular failure not due to dehydration the ætiological factors were toxæmia and anoxæmia. Once the condition of peripheral vascular failure had developed, with the tardy peripheral circulation and the falling blood pressure, the medical practitioner knew how difficult it was to relieve the condition. Such drugs as adrenaline gave only fleeting benefit and must be exhibited with great care. Dr. Hetzel himself favoured pituitrin in smaller doses and given more frequently than Dr. Gartrell advocated. This drug did act on the peripheral vascular field without raising the blood pressure much, if at all, and it could be of help. Dr. Hetzell's experimental observations on human beings led him to the belief that pituitrin did not raise the blood pressure.

Anoxæmia played a large part in the causation of peripheral vascular failure and so the continuous giving of oxygen was advocated before the patient showed signs of cyanosis, especially if he was past middle age and had a definite cardiac lesion.

As regards toxæmia, although antitoxin was used in diphtheria, in Australia anti-pneumococcal serum was not used for Type I pneumonia in order to combat the toxæmia.

There existed a non-specific measure which was of great value, not only in prophylaxis by supporting the peripheral vascular field, but also in treating the failure if it developed. Dr. Hetzel referred to hydrotherapy. Tepid sponging, without distressing the patient, improved the tone of the peripheral vessels and supported the circulation generally. Dr. Hetzel endorsed its use both as a prophylactic and as a therapeutic measure. In these conditions Dr. Hetzel agreed with Dr. Gartrell that definite indications for its exhibition should be awaited before digitalis was used; for by such measures as giving strophanthin intravenously, or digoxin, the patient could be brought under the influence of the drug rapidly.

Dr. Hetzel next referred to nursing. Since pneumonia had been taken as the disease from which deductions could be drawn as to the effect of toxæmia in the circulation and the necessity for supporting it, did they perhaps insist too much on Fowler's position for patients with pneumonia? Was this position adjuvant to the patient's circulation and to his rest? Might they not be exhausting him unnecessarily by making it a routine measure and by putting the patient back into it when he slipped down or seemed to become uncomfortable? Dr. Hetzel had been interested in this aspect of the subject for years and was gratified to find during his recent visit to America that at the Johns Hopkins Hospital and other institutions the patient with pneumonia was permitted to take up the position in which he was most comfortable. Quite a large percentage of the patients seemed to prefer to lie on the side with one or two pillows. Necessarily this position would vary according to the factors involved, whether respiratory pain, cardiac embarrassment, or other factors.

As regards brachycardia, Dr. Hetzel thought that in early convalescence the persistent brachycardia must be considered as evidence of cardiac exhaustion, if not of a dual cardiac dilatation; prolonged rest and the exercise of care on the patient's return to activity were essential.

In conclusion, Dr. Hetzel thanked Dr. Gartrell for his lucid exposition of a difficult subject and for the fact that he had not attempted to load his paper with too much detail.

DR. D. R. W. COWAN asked whether oxygen administration was, in Dr. Gartrell's experience, of value if given by a nasal catheter, and whether it would be advisable to administer oxygen earlier than was usual in some cases.

DR. F. H. BEARE mentioned the low mortality rate at Northfield from cardiac failure in diphtheria and compared it with the higher rate in pneumonia. Was this disparity because the former disease fell on virgin soil? He also quoted from the Johns Hopkins Hospital reports in which myocardial changes were said to have been found in patients who had died from diphtheria during the first week from the onset.

DR. JOHN HAYWARD asked if Dr. Gartrell thought there was any great risk or benefit attached to the creation of an artificial pneumothorax in lobar pneumonia.

DR. C. SANGSTER asked Dr. Gartrell's opinion on the value of "Coramine" as a cardiac stimulant.

DR. GUY LENDON remarked on the fact that venesection at one time was universally practised in the treatment of pneumonia and asked Dr. Gartrell what was his experience of its value.

DR. IAN HAMILTON warmly agreed with the suggestion of operation and cooperation with the surgeon in hyperthyreoidism.

DR. H. T. ILLINGWORTH wished to know if Dr. Gartrell had used "Carbogen" in the treatment of pneumonia.

Dr. Gartrell, in reply, said that oxygen administration by means of a tent was the ideal method, but that tents were not usually available. Provided the flow through the catheter was kept going at the proper rate, continuously day and night, the effects were undoubtedly beneficial. This view was supported by Professor Wynn in *The British Medical Journal* of January 11, 1936. Although oxygen saturation of the blood was normal in pure heart failure, this was not always the case when heart failure occurred in pneumonia. If the heart began to fail, its immediate response was to dilate, so lengthening the muscle fibres, in other words, increasing the length over width ratio. This proportionately increased the surface and so increased the facility for chemical interchange. Thus the heart could deal with the extra work, but in order to do so it was consuming more oxygen per unit of work. Hence oxygen might be expected to be of assistance in these circumstances. Indeed, it was known that anoxæmia sometimes precipitated heart failure. It was quite conceivable that although early administration of oxygen was useless in most cases it was of service in those patients whose hearts were about to fail.

Referring to Mackenzie's statement concerning sinus arrhythmia, Dr. Gartrell said that Mackenzie subsequently retracted. As regards the position in which the patient was nursed, Dr. Gartrell was in entire agreement with allowing him to have just as few pillows as were consistent with his comfort. When peripheral failure was causing symptoms, the recumbent position was obviously to be desired.

In patients with diphtheria and peripheral failure a binder was sometimes used to minimize stagnation in the visceral vascular field. However, when cardiac failure occurred the patient's venous pressure rose and led to dyspnœa, in which condition it was necessary for him to be propped up. This discomfort was due mainly to reflex effects through the vagus owing to some blood leaving the abdomen and going to aid the pulmonary congestion when the patient was lying flat. Some thought it was in part due to increased venous pressure in the region of the respiratory centre, which was relieved, of course, when the sitting position was adopted. Dr. Gartrell agreed that the effect of adrenaline was evanescent and uncertain. Pituitrin had been found to exert a more potent effect when a 50% solution of glucose had been injected into a vein. However, it was rather late to get good results from either drug when toxic damage had occurred. The remedy lay in treating the diseases themselves by specific measures when this was possible. This specific treatment probably was the cause of Dr. Beare's low mortality rate at Northfield in cases of diphtheria. As treatment by this means was so much more certain in diphtheria than it was in pneumonia, this factor was probably more active in producing the difference between the figures for the two diseases. Actually Dr. Beare's virgin soil was less experienced in resisting the insidious attacks of bacilli, and this might counter the beneficial effects of its purity and youthful vigour.

The findings in the first week of the disease were inconsistent in different series and, what was more, the manner of death during that period was quite different from that of true heart failure. There was no congestive failure with enlarged liver and venous congestion, but the patient was pale, clammy, "shocked" and faint, with a rapidly diminishing blood pressure and a pulse that gradually or suddenly grew smaller until it disappeared. This was death from peripheral failure.

In reply to Dr. Hayward, Dr. Gartrell said that the good results of the induction of a pneumothorax in lobar pneumonia probably were greatest when distressing pain was abolished by the separation of the serous layers by the air. Pain was a recognized cause of shock and therefore might conceivably aggravate a condition akin to shock, namely, peripheral failure. Pain also led to lack of rest, to increased emotional strain and so to increased cardiac work. It also interfered with diastolic filling by hampering respiration. As the amount of lung involved was not the main factor influencing prognosis in this disease, it was unlikely that the introduction of 200 cubic centimetres of air was going to influence the patient adversely, as it was spread out in a thin layer.

In reply to Dr. Sangster, Dr. Gartrell said that experimental evidence showed that "Coramine" was a valuable respiratory stimulant, but as a cardiac stimulant it was entirely impotent. Professor Hicks had shown results of this quite recently, and cardiologists generally were of the same opinion.

In reply to Dr. Guy Lendon, Dr. Gartrell said that venesection used to be employed far too frequently, but it later fell into undeserved disrepute. It was now recognized as a valuable measure when certain indications were present. Dr. Gartrell's view of its use in pneumonia was that when the heart was suffering from toxic damage and beginning to find its load too great—when it was beginning to fail—venesection acted by reducing the work of the heart. Certainly this was only a temporary effect, but it did give the heart an opportunity of regaining some of its tone, and as it was known that patients who survived had undamaged hearts, it was clear that venesection meant playing for time and that it might tide the heart over the most dangerous period until the toxæmia had lessened.

Dr. Hamilton had touched on a vital spot when he referred to cooperation with the surgeon and operation in cases of hyperthyreoidism. Early operation, with its mortality of only 1%, was a sound method of preventing heart failure when reasonable medical treatment was not sufficient. In the presence of failure the mortality varied from 4% to 20%, which was a much better figure than that for medical treatment of hyperthyreoidism with heart failure. Cardiac failure was not only not a contraindication for operation, but an urgent indication for it.

Regarding Dr. Illingworth's question, Dr. Gartrell said that "Carbogen" was undoubtedly of value in some cases of pneumonia, but was indicated only in the presence of respiratory failure.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Melbourne Hospital on May 20, 1936. The meeting took the form of a series of clinical demonstrations. Part of the report of this meeting was published in the issues of June 20 and July 11, 1936.

Congenital Pulsating Exophthalmos.

DR. T. FRANK showed a male patient, twenty-six years of age, who had reported on November 27, 1935, complaining of a pain in the back following an accident three weeks previously. He also asked advice as to the movements of his left eye, which had been causing him embarrassment in company; he stated that these eye movements had been present since birth, but that his eyesight was very good. On examination there was definite proptosis and pulsation of the left eye, synchronous with the pulse beat. Obliteration of the common carotid artery stopped the pulsation of the eye, but the eye movements and the vision were good. A small angioma was present on the margin of the left upper lid. Examination showed the veins in the left fundus to be slightly fuller than those in the right.

A radiological examination revealed that the left orbit was enlarged in comparison with the right, the ethmoids were narrowed, the bones of the roof, floor and sides of the orbit were so thinned out that their markings were completely absent; the left optic foramen appeared to be narrowed, but, owing to the thinning of the bone, it was difficult to demonstrate the foramen. In addition, the *sella turcica* was increased in size and much deeper than usual. The blood did not react to the Wassermann test. The ear, nose and throat specialist had reported a deviation of the septum to the left and some infection in the ethmoid region.

In this case a diagnosis of congenital pulsating exophthalmos due to an angioma, intracranially situated, was made; this was also suggested by the angioma on the left upper eyelid.

Dr. Frank said that over six hundred cases of pulsating exophthalmos had been recorded in the literature; the majority were due to a communication between the internal carotid artery and the cavernous sinus, and at least 77% of the cases were the direct result of trauma (Terry and Mysel). The pulsation was explained by the fact that the anastomosis between the artery and the veins caused a rise of pressure, which thus carried the arterial pulse to the eye. Reid, in *Archives of Surgery*, 1925, stated that the great majority of angiomata were composed of dilatations of the smallest arteries and veins which communicated freely with each other. Moreover, this type exhibited no tendency to undergo malignant changes. The thinness of the orbital walls and the deepening of the *sella turcica* in this case were most probably the result of the angioma.

It had been decided not to commence any treatment in this case at that time; should impairment of vision or increased proptosis occur, then surgical treatment might have to be undertaken. Reid had recommended ligation of the common and external carotid arteries and also the supraorbital vein; other authorities had recommended ligature of the internal jugular vein in addition. Such complications as sudden death or hemiplegia might follow ligature of the common carotid artery, and in this case, at present, the risk of an operation was not justified.

Pneumonokoniosis and Leontiasis Ossea.

Another patient shown by Dr. Frank was a male, sixty years of age, who had worked as a quartz-miner for

fourteen years and who had reported to the out-patient department on September 23, 1935. He had stated that he had a cough with expectoration of sputum over a period of three years and that he lacked energy and had become dyspnœic on exertion. His sputum had been blood-stained on several occasions, and during the past twelve months he had lost 12·6 kilograms (two stone) in weight; the left side of his face had been swollen for many years, but had not enlarged recently.

Examination showed a definite overgrowth of the left maxilla in all directions, and the lungs showed pulmonary fibrosis, which was associated with emphysema; in addition, the heart was slightly enlarged and the arteries were thickened. Radiological examination of the skull revealed *leontiasis ossea*, with a definite overgrowth of the left maxilla; and a similar examination of the chest showed a well-defined pneumonokoniosis with massive lung density of the upper half of both lungs; the extreme apices were clear, with no characteristic tuberculous involvement. The sputum contained no tubercle bacilli and the blood did not react to the Wassermann test.

From the clinical and radiological examinations a diagnosis of pneumonokoniosis and *leontiasis ossea* was made. This case was shown to illustrate the appearance of the face in this rare bone disease. The name leontiasis was given by Virchow because the appearance of the face resembled the leontiasis of leprosy. It might occur at any age, but usually in the young; least frequently it occurred in the first decade. Knaggs had described two types of this condition: (i) a chronic periostitis spreading slowly from bone to bone of the face and skull; (ii) a diffuse osteitis of the bones of the face and skull, which might be generalized, localized or circumscribed. Dr Frank said that this patient probably had a localized form of the second group, limited mainly to the left maxilla. The ætiology of the disease was unknown and many people held different views; some regarded it as the result of a creeping infection from the nasal sinuses, others as the result of trauma and due to a congenital predisposition of the periosteum and bone marrow to grow. Others considered that *leontiasis ossea* was not a pathological entity, but either a manifestation of Paget's disease or of von Recklinghausen's disease (*osteitis fibrosa cystica*); there was no known treatment for it.

Rheumatic Carditis and Mitral Stenosis.

DR. LESLIE HURLEY showed a female patient, twenty-six years of age, who had reported complaining of dyspnœa on exertion, which had been increasing, and loss of voice for four months. The patient gave a history of an illness with acute rheumatism at thirteen years of age, when she spent three months in bed. On examination of the heart the apex beat in the left sixth intercostal space was 13·75 centimetres (five and a half inches) from the mid-line; the first apex sounds were snapping and the second pulmonary sound was reduplicated and accentuated; there were also systolic and diastolic murmurs at the apex, and a presystolic murmur at a small area inside the apex; the heart also was greatly enlarged to the left and somewhat to the right. Gallop rhythm was also present at the apex beat. The right lung was dull at the base, with diminished vesicular murmur; the abdomen was normal, and no abnormality was detected in the central nervous system. The left recurrent nerve of the larynx was paralysed. A radiographic examination of the chest showed an enlargement of the left auricle with a projecting conus of the pulmonary artery; and a barium meal test of the œsophagus demonstrated the enlargement of the auricle. The diagnosis was rheumatic carditis, mitral stenosis, with left recurrent nerve paralysis.

The following points of interest were discussed by Dr. Hurley in connexion with this case: (*a*) The presence of a left recurrent laryngeal paralysis was due to the great enlargement of the left auricle. The differential diagnosis from aneurysm, pericardial effusion and pleural effusion was also discussed. It was pointed out that the left auricle enlarged backwards and to the right, and therefore might cause a considerable area of dulness in the chest. (*b*) Radiographic plates were also exhibited, illustrating the enlargement of the left auricle; the characteristic cardiac outline was that seen in most cases of mitral stenosis. (*c*) The different types and significance of gallop rhythm were discussed. (*d*) Some electrocardiograms illustrating the preponderance of the heart were shown.

Syphilitic Aortitis and Neuro-Retinitis.

Dr. Hurley also showed a male patient, fifty-two years of age, with syphilitic aortitis and neuro-retinitis. The patient had complained of misty vision for three weeks and had had a past history of chancre on the penis one year previously, followed a month later by a rash on the body and ulcers in the mouth. Six months previous to the meeting he had had hepatitis, probably due to arsenical treatment or partly to bismuth and the syphilitic infection.

On examination of the heart the apex beat in the sixth intercostal space was found to be 10·0 centimetres (four inches) from the mid-line; and there was pulsation in the second right intercostal space. There was a systolic and diastolic bruit at the aortic area, conducted down the left side of the sternum, and the aortic mid-line sound was loud and ringing. The systolic blood pressure was 165 and the diastolic pressure was 90 millimetres of mercury; no other abnormality was detected in the lungs or abdomen. The Wassermann test gave no reaction at the time of the meeting, but had given a positive reaction ten months earlier. There was a blurring of the retina and optic disks, and vitreous opacities were present in both eyes.

The following points of interest were discussed in regard to this case: (*a*) The development of signs of aortitis within one year of the primary infection. The possibility of a previous syphilitic infection was also discussed, but was considered to be most unlikely. (*b*) The causes and treatment for jaundice in patients under treatment for syphilis. (*c*) The occurrence of active manifestations of syphilis in the presence of a failure of the blood to react to the Wassermann test.

Dr. Hurley also showed some radiographic films demonstrating the enlargement and lengthening of the aorta; the appearances seen in this case were then contrasted with those found in mitral stenosis.

Innominate Aneurysm.

DR. G. R. A. SYME showed a female patient, thirty-five years of age, who had complained of hoarseness for seven weeks and of some tingling down the right arm and up the right side of the neck. On examination Dr. Syme had found a pulsating expansile swelling just above the sterno-clavicular joint on the right side and extending down towards the *manubrium sterni*, which the patient said had been present for twelve years, but had recently become slightly more prominent; there was also a small hairy patch over the tumour. No sensory or muscle changes were found in the right arm; but the radial pulse at the left wrist was much weaker than that on the right side, though it improved on elevation of the arm. The blood pressure was equal in both arms; the heart was normal and the carotid pulses were equal. Horner's syndrome could not be demonstrated, but the right vocal cord showed early abductor paralysis. A blood specimen had been sent for a Wassermann test, but the report had not been returned at the time of the meeting. The cervical spine had been examined by X rays and the absence of a cervical rib was established.

In the diagnosis Dr. Syme had considered that the condition might be a subclavian aneurysm caused by a cervical rib, a right common carotid artery arising from the left side of the aortic arch and presenting very superficially, a low carotid aneurysm, or an innominate aneurysm. The skiagram had excluded the presence of a cervical rib, though there was still a possibility that a fibrous band was present. Dr. Syme showed a skiagram taken after an injection of "Thorotrast" into the swelling at the sternal notch with the right carotid and the right subclavian arteries compressed. Though the film was inconclusive, it showed the presence of a normal right common carotid artery; and the innominate artery appeared to be enlarged.

The nervous symptoms were obviously due to pressure on the brachial plexus and recurrent nerve, but no definite explanation could be offered for the weakened left pulse. Dr. Syme thought it probable that there had been a congenital weakness of the wall of the artery, as there was no evidence of syphilis or of arteriosclerosis. With reference to treatment, he proposed to watch to see whether the left cord would compensate for the paralysis of the right, because the huskiness was the only symptom of which the patient was complaining, and the risk of hemiplegia resulting from ligation was considered to contraindicate immediate operative interference.

Post-Operative Progressive Gangrene of the Skin.

Dr. Syme also showed a male patient, fifty years of age, with post-operative progressive gangrene of the skin. The patient had been admitted to hospital with an acute gangrenous appendicitis and the appendix was removed through a muscle-splitting incision. A small abscess at the tip of the appendix burst during removal and the peritoneal cavity was drained. Two days after the operation the wound round the tube developed cellulitis and all the sutures were removed and the condition was treated by foments and dressings of hypertonic saline solution. At the end of a week the condition had gradually spread and involved an area of four square inches around the site previously occupied by the tube. Towards the centre, sloughing, gangrenous skin and subcutaneous tissues resembled a carbuncle; outside this was an area, one inch wide, of bright hyperæmia, which was not painful. There was practically no general reaction, and the patient's temperature had not risen above 37·2° C. (99° F.).

Dr. Syme said that every type of antiseptic was tried, but all without effect; the condition slowly progressed until at the end of a month it occupied about three-quarters of one square foot of the abdomen wall. Antiseptics having failed, the area was excised, the incision commencing a quarter of an inch outside the hyperæmic area and extending down to the muscular aponeurosis. This left a large raw area, which granulated well; it was grafted by Thiersch's method a fortnight later, and the graft took completely. There was no further trouble with the spreading gangrene. Bacteriological examination revealed a microstreptococcus in the hyperæmic area and several different organisms in the gangrenous area, of which Dr. Lucy Bryce thought a hæmolytic staphylococcus was the most pathogenic. Dr. Syme considered that neither of these organisms by itself was very pathogenic, but the progressive gangrene was caused by a synergistic action of the two bacteria. He showed photographs of the condition before and after operation and remarked that the condition was almost invariably associated with operations involving the intestinal tract.

A Late Rupture of the Extensor Pollicis Longus.

Another patient shown by Dr. Syme was a female, thirty years of age. Seven weeks before reporting, the patient had sustained a Colles's fracture without displacement, which had been treated on a splint for four weeks. She then returned to work, and seven weeks later, while using her thumb, she had felt a sudden severe pain over the site of the fracture and was unable to extend the distal phalanx; she had had slight pain over the site of the fracture all the time. Dr. Syme said that on examination a gap could be felt in the course of the *extensor pollicis longus* and complete inability to extend the distal phalanx of the thumb was demonstrable. At operation the tendon was found to be completely divided and the ends were very frayed. These findings were suggestive of continuous friction from a fragment of projecting bone, but no such projection was found. The proximal end of the tendon was transplanted from its groove to the radial side of Lister's tubercle, and by hyperextending the thumb the tendon was sutured with silk and a sheath was refashioned. Active movement of the terminal phalanx was commenced in forty-eight hours, and the movement was gradually increased until after ten days the hyperextension splint was removed. At the time of the meeting, ten months after operation, the patient exhibited perfect function and power in every respect. Dr. Syme said that other operations used for this injury included transplantation of the tendon of the *extensor indicis proprius* to the distal end of the severed tendon or the use of a piece of *fascia lata* as a graft.

Carcinoma of the Ureter.

Dr. Syme also showed a female patient, sixty-seven years of age, who some seven weeks earlier had been wakened with acute colicky pain in the right loin, moving down to the right groin. The pain lasted several hours, and though there had been no recurrence, she still had a dull ache in the loin. The first specimen of urine passed after the pain had been brightly blood-stained and every subsequent specimen had contained blood. She had also developed frequency of micturition by day and by night. In the past she had not had any ill-health, and the urological history was quite clear. On examination it was noted that she was a thin, anæmic woman, and the right kidney was readily palpable and enlarged. In other respects, including a skiagram of the chest, there were no abnormal findings. A provisional diagnosis of a papillary carcinoma of the renal pelvis with a secondary hydronephrosis was made, because the kidney felt like a hydronephrotic kidney, being normal in shape and freely mobile. By a cystoscopy it was seen that the bladder was of normal appearance, and dye appeared definitely from the left ureter in four minutes, but no dye or efflux came from the right ureter. A ureteric catheter could not be passed more than 3·75 centimetres (one and a half inches) up the right ureter, and it was thought to be blocked by a mucous fold.

The examination was repeated on two occasions with similar results, and sodium iodide could not be injected up to the kidney pelvis. "Uroselectan" was not used, as it had to be provided at the patient's expense, and Dr. Syme considered that the diagnosis of carcinoma of the kidney was justifiable. The kidney was explored and a large hydronephrosis was found; there was a definite kink of the ureter in an adherent mass 3·75 centimetres (one and a half inches) down from the pelvis. The mass was freed from the ureter, but the ureter below the kink was not explored. Subsequent to the operation the hæmaturia continued, and at further cystoscopic examinations a blood clot was seen to be coming from the right ureter. A tentative diagnosis of carcinoma of the ureter was made and it was decided to remove the ureter. This was done through a transperitoneal approach and the dilated ureter and small tumour in its lumen, 3·75 centimetres (one and a half inches) from the bladder, were removed. The post-operative course was uneventful.

Dr. Syme exhibited the specimen, a section of which showed papillary carcinoma. Only sixty-one cases had been reported and in only three or four of them had a correct pre-operative diagnosis been made. After the first operation the fact that a hydronephrosis could cause such profuse hæmaturia had been noted with surprise, and subsequent events had shown that the surprise was justified.

Public Health.

THE WOMEN'S WELFARE CLINIC OF THE MELBOURNE DISTRICT NURSING SOCIETY.

THE latest report of the medical officer in charge of the Women's Welfare Clinic of the Melbourne District Nursing Society has been received. This is one of the pioneer birth control clinics in Australia. It was started about eighteen months ago. Free instruction and suitable supplies have been given, after examination, to women in need of contraceptive advice on medical grounds. Very complete records are kept. The cooperation of the patients has been excellent, and their appreciation of the service has been a source of much satisfaction to those in charge.

The Melbourne District Nursing Society has been carrying on domiciliary nursing in the homes of the poor people of Melbourne and suburbs for the past fifty years. On the midwifery side about five hundred cases are attended each year; and nearly 1,000 "general" visits are paid every week. Expectant mothers attend the antenatal clinic or the Prahran Health Centre. The nature of the work makes the contact with patients much more personal than is the case in hospital practice. The tragedy of pregnancy in the medically unfit is most forcibly brought before the medical officers. Their views on this subject were placed before the Committee of the Melbourne District Nursing Society and permission was given for the establishment of a welfare clinic, where contraceptive instruction could be given to patients from antenatal clinics for health reasons. The cost of the clinic (£40 to date) has been covered by special donations, and no charge has been made to the patients.

The report of the medical officer to the clinic is as follows:

The patients who have sought instruction at the Welfare Clinic since it was started on October 17, 1934, number 130. The following list includes those cases referred to in the last report, which was to June 27, 1935. Of the 130 women who have attended, two came because of sterility. They were examined and referred to the Women's Hospital for further investigation and treatment. In every case where instruction in contraception was given very definite medical indications were present, as follow:

General ill-health due to rapidly repeated pregnancies	41
Various forms of heart disease	22
Chronic Bright's disease	11
Neurasthenia, threatened mental derangement and other neuroses	8
Recent extensive pelvic operations	5
Thyreoid disease, myocarditis	4
Secondary anæmia, debility	3
Recent eclampsia (renal function very poor)	3
Diabetes with other serious disabilities	3
Pulmonary tuberculosis	2
Chronic pyelitis	2
Abscesses of breasts, chronic ill-health	2
Contracted pelvis (Cæsarean section and several tubal inductions)	2
Uterine prolapse and cardiac disease	2
Ill-health following recent septic abortion	2
Rectocele and cystocele (awaiting operation)	2
Active syphilis (with three miscarriages)	1
Deformed skeleton (crippled by poliomyelitis), difficult labour	1
Chronic rheumatism, recurrent pleurisy	1
Strong family history of pseudo-hypertrophic paralysis (9 boys affected), husband totally blind	1
Emaciation, neurosis, rapidly repeated pregnancies	1
Incontinence of urine following bladder injury	1
Chronic nephritis, syphilis	1
Very persistent trichomonas vaginitis	1
Chronic mastoid suppuration (awaiting operation)	1
General ill-health—retroverted uterus (awaiting operation) strong family history of insanity	1
Sequelæ of appendiceal abscess (to have another operation)	1
Tubercular peritonitis (pensioner)	1
Anæmia and debility, recent operation for ectopic pregnancy	1
General ill-health; husband an invalid pensioner (tuberculosis)	1
	128

It will be noticed that by far the largest group is the first on the list—"General ill-health due to rapidly repeated pregnancies". This may sound vague, but in each case the need for advice was most urgent. Constitutionally these women are not fit for endless child-bearing. Most of the patients in this group stated that they were "very run down" or that they suffered from "nerves". Many were on sustenance, while in other cases the husband was an invalid pensioner. In one case the patient had been in hospital for nine weeks following septic abortion. Blindness supervened and both eyes had to be removed. Fifteen women belonging to this group have had ten or more children, the largest family being fifteen children.

It is of interest to record that one patient who was asked to attend the clinic could not do so on account of the illness of her child, who died after a few months. Meanwhile the mother became pregnant. In desperation she induced an abortion. She became septic and died in the Women's Hospital. This could have been prevented by timely instruction at our clinic.

The ages of the women who have been given instruction varies from 21 to 47 years, the average age being a little less than 33 years.

Six children was the average number born to each patient who attended the clinic.

The contraceptive method advised is simple, harmless and effective. No known method gives 100% of success with everyone who employs it. The human element must be taken into account. Out of 128 cases there were three true failures. Three other cases were actually a few weeks pregnant when they first visited the clinic. One woman neglected to use any contraceptive method at all and promptly became pregnant. One woman desired another baby, so discontinued using any birth control methods. The remaining two who have become pregnant have not yet been interviewed.

It appears, however, that we have achieved approximately 97% of successes, which is as good as any of the leading clinics in England. With a slightly improved method it is hoped that this percentage will be still higher.

In conclusion it may be said that all who are directly associated with the Women's Welfare Clinic feel that the work is of very great importance, both medically and socially, and we are grateful to the Committee of the Melbourne District Nursing Society for allowing us to use the antenatal department for this very necessary work.

VICTOR H. WALLACE.

105, Collins Street,
Melbourne.

Correspondence.

PROTECTIVE FOODS.

SIR: I notice in the interesting and valuable article entitled "The Modern Outlook on Nutrition" (THE MEDICAL JOURNAL OF AUSTRALIA, June 20, 1936) that Dr. Noel M. Gutteridge includes meat among the protective foods. Sir Robert McCarrison, who is, I suppose, our greatest living authority on diet, dissents from this inclusion. In his lecture, reported by *The British Medical Journal* of February 29, 1936, page 430, column 2, paragraph 4, the following statement occurs: "He rather dissented from the inclusion in a recent authoritative report of meat as among the 'protective foods'. It was probably so included because it was a rich source of good protein and of vitamin B_2; but it was poor in certain other essentials, and by naming it 'protective' the impression was created that health depended on its inclusion in the diet, which it did not."

Yours, etc.,

INA M. HOECKER.

Darlinghurst,
Sydney,
June 24, 1936.

The Royal Australasian College of Surgeons.

MEETING OF BOARD OF CENSORS.

THE next meeting of the Australian Board of Censors of the Royal Australasian College of Surgeons will be held in Melbourne on Saturday, August 22, 1936. Candidates who desire to present themselves at this meeting should apply to the Censor-in-Chief for permission to do so, on or before July 31, 1936. The appropriate forms are available at the College, Spring Street, Melbourne, and at the offices of the various State secretaries.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on July 6, 1936.

The following degree was conferred: Bachelor of Surgery (B.S.), Neil Acheson Gordon.

The Diploma in Psychiatry was awarded to William Ernest Audley, M.B.

The Diploma in Tropical Medicine was awarded to John Howell Blakemore, M.B., Charles Mervyn Deland, M.B., Abraham Fryberg, M.B.

An amount of £2,000 was received from the executors of the will of the late Miss Maria Fanny Sanders (The Perpetual Trustee Company Limited, Messrs. J. R. Spencer and A. E. Buzacott). The income from this legacy is to be used for the establishment of a scholarship, to be called the "Sister Sanders Scholarship", for research work in the prevention of disease in children. The Faculty of Medicine has been asked to draw up suitable conditions for the award of the scholarship.

A former University bursar, in a letter addressed to the Senate, expressed her appreciation of the bursary assistance which had been given to her to enable her to obtain her degree. This bursar stated that she wished to repay the amount to the University by annual instalments of £5. The Senate has expressed its appreciation of the action taken.

The following appointments were made: Dr. N. R. Wyndham as Part-Time Demonstrator in Physiology; Dr. W. S. Dawson as Lecturer in Psychiatry for the year 1936.

On the recommendation of the Professorial Board, the Senate decided not to place any general age restrictions on students entering the University. The Senate has previously decided that no student under the age of seventeen years should be admitted to the Faculty of Law.

On the recommendation of the Faculty of Veterinary Science, the Walter and Eliza Hall Veterinary Science Research Fellowship was awarded to Mr. H. B. Carter, B.V.Sc. Mr. Carter graduated in 1933. Under the terms of his appointment he is to carry out research in connexion with factors associated with fleece density.

The following conditions for the award of the Earle Page Constitutional Prize were approved:

1. The prize to be known as the Earle Page Constitutional Prize.
2. The prize to be awarded every two years.
3. The value of the prize to be fixed at £8.
4. The prize to be open to undergraduates and graduates of not more than eighteen terms' standing.
5. The prize to be awarded for an essay upon a subject of constitutional, political or administrative interest.
6. (*a*) The first award to be made in 1937; (*b*) the subject for 1937 to be "The Financial Relations of the Commonwealth and the States".

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act* 1912 *and* 1915, of New South Wales, as duly qualified medical practitioners:

Hogg, Geoffrey Charles Huxtable, M.B., B.S., 1933 (Univ. Sydney), c.o. Commonwealth Bank, Australia House, Strand, London.

Sweeting, John Stretch Deane, M.B., B.S., 1934 (Univ. Melbourne), Women's Hospital, Crown Street, Sydney.

Additional diploma registered:

Shannon, James Robert, D.P.H. (Univ. Sydney).

TASMANIA.

The undermentioned has been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Turnbull, Reginald John David, M.B., B.S., 1933 (Univ. Melbourne), Public Hospital, Launceston.

QUEENSLAND.

The undermentioned have been registered, pursuant to the provisions of *The Medical Acts*, 1925 *to* 1933, of Queensland, as duly qualified medical practitioners:

Adam, John Robert, M.B., B.S., 1936 (Univ. Melbourne), Brisbane.

Anderson, Bruce Hunter, M.B., B.S., 1936 (Univ. Melbourne), Brisbane.

Beaumont, John Richard Besnard, M.B., B.S., 1933 (Univ. Sydney), Stanthorpe.

Hecker, Cyril Ferdinand, M.R.C.S. (England), L.R.C.P. (London), 1929, Stanthorpe.

O'Toole, Cyril Paul, L.L.M., R.C.P. and S., 1927 (Ireland).

Scott, Henry Maynard, M.B., Ch.M., 1924 (Univ. Sydney), Dirranbandi.

O'Connor, Joseph Lionel, M.B., B.S., 1931 (Univ. Melbourne), Brisbane.

Robertson, Arnold William, M.B., B.S., 1926 (Univ. Sydney), Brisbane.

Weedon, Dacre de Vaux, M.B., 1926 (Univ. Sydney), Brisbane.

Additional diploma registered:

Burne, Alfred Rainald Keith, F.R.C.S. (Edinburgh), 1930.

Obituary.

BEDLINGTON HOWEL MORRIS.

WE regret to announce the death of Dr. Bedlington Howel Morris, which occurred on July 10, 1936, at Adelaide, South Australia.

Books Received.

LEAGUE OF NATIONS: QUARTERLY BULLETIN OF THE HEALTH ORGANIZATION; Volume V, Number 1, March, 1936. Geneva: Publications Department of the League of Nations; Sydney: The Times Limited. Royal 8vo, pp. 209. Price: 2s. 6d. net.

THE HYGIENE OF THE CHANGE IN WOMEN (THE CLIMACTERIC), by I. E. Hutton, M.D.; 1936. London: William Heinemann (Medical Books) Limited. Crown 8vo, pp. 119. Price: 5s. net.

Diary for the Month.

JULY 21.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 21.—Tasmanian Branch, B.M.A.: Council.
JULY 22.—Victorian Branch, B.M.A.: Council.
JULY 23.—New South Wales Branch, B.M.A.: Clinical meeting.
JULY 24.—Queensland Branch, B.M.A.: Council.
JULY 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 30.—New South Wales Branch, B.M.A.: Branch.
JULY 30.—South Australian Branch, B.M.A.: Branch.
AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 4.—Tasmanian Branch, B.M.A.: Council.
AUG. 5.—Western Australian Branch, B.M.A.: Council.
AUG. 5.—Victorian Branch, B.M.A.: Branch.
AUG. 6.—South Australian Branch, B.M.A.: Council.
AUG. 7.—Queensland Branch, B.M.A.: Branch.
AUG. 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 11.—Tasmanian Branch, B.M.A.: Branch.
AUG. 14.—Queensland Branch, B.M.A.: Council.
AUG. 18.—New South Wales Branch, B.M.A.: Ethics Committee.
AUG. 18.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments.

Dr. R. W. Dungan has been appointed a Certifying Medical Practitioner at Yarraville, Victoria, for the purposes of the *Factories and Shops Act*, 1928.

* * *

Dr. J. L. O'Connor has been appointed a Quarantine Officer under the *Quarantine Act*, 1908-1924.

* * *

Dr. H. W. S. Hambrett has been appointed Medical Officer, Office of the Director-General of Public Health, New South Wales.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xv, xvi, xvii.

DEPARTMENT OF HEALTH, SYDNEY, NEW SOUTH WALES: Medical Officer.

DEPARTMENT OF PUBLIC HEALTH, PERTH, WESTERN AUSTRALIA: Medical Officer.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.

IPSWICH HOSPITALS BOARD, IPSWICH, QUEENSLAND: Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Registrars.

REPATRIATION COMMISSION, ADELAIDE, SOUTH AUSTRALIA: Medical Officer.

ROYAL MELBOURNE HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.

THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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